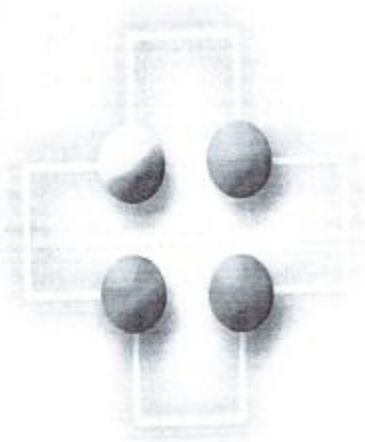
1

MEDICAL HISTORY



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INTRODUCTION

In Iraq, there are three types of clinical examination; the long case, the short case, and the OSCE examinations. Undergraduate and postgraduate medical students may face them at the time of clinical training. Indeed additional modes of examination are sometimes adopted to assess the ability of the students to elicit abnormal finding e.g. slide show and oral examination. The candidate's duty in long case exam is to perform a thorough history and physical examination. There is quite an art in presenting a case concisely and clearly. The examiners have no time to waste, and hesitant and long winded presentations are tedious. Thus the students should practice every day case presentation to be a good historian.

The physical examination on the other hand is usually performed under four headings. These are; the general examination, vital signs measurements, systemic examination and sometimes special examination for common surgical presentation. This rule is applied for all undergraduate medical students but not the third medical class. For whom history and general examination as well as vital signs measurements are quite sufficient. Older students should be ready for any physical examination according to their clinical course. Of course, in clinical pediatrics, growth chart measurements are invariably added. Like wise in clinical obstetrics, obstetrical examination of the abdomen is eventually added.

In the short case examination, the senior examiners usually prepare three cases, or to a lesser extent two cases, each of which has valuable medical importance. The candidates here should listen carefully to the instructions of the examiner with regard to the part or system to be examined and obey them implicitly. The major skill required is of course the ability to elicit physical signs correctly but other factors such as fluency in case presentation and clinical judgment in the interpretation of the physical signs elicited are also important.

An OSCE is an Objective Structured Clinical Examination. It is designed to reduce some of the problems associated with the traditional short and long case examinations. In typical OSCE examination, students rotate around a circuit consisting of a number of stations lasting a short, fixed time. In that way, all students are asked the same questions at identical stations.

In any clinical examination, the followings are recommended:-

1. The candidates should be fully equipped.
2. The history is taken as well as the examination is performed always on the right side of the patient's bed.
3. The language used is the high level UK English language (no way for colloquial or slang words), but when Arabic language seems to be appropriate, a permission should be requested saying for example (have I an excuse to talk in Arabic language?).
4. At the end of examination the candidates should re-cover the part being exposed and should be grateful for the patient.

Ethical issue

Optimal respect for both the examiner and the patient is first and forever, always more important than the student's scientific level. The patients' suffer should be sought however minor. Indeed the patient will only provide correct answers provided you gently ask the right question. Always assume that the patients are able to fully understand the presentation whatever their educational level. The students, therefore, should assume no harmful or flippant comments. More over, the students should not tell the patient about his/her illness as some facts would be too terrible a burden for him/her.

Equipments

The equipments needed vary according to grade of the medical students. Third class medical student should have a stethoscope, thermometer and a sphygmomanometer. Older student should have the following:-

1. Stethoscope.
2. Sphygmomanometer.
3. Tendon hammer.
4. Wooden tongue depressor.
5. Measuring tape.
6. Thermometer.
7. JVP ruler.
8. Pins, red-headed pin, and wooden pins.
9. Pen torch.
10. Cotton wool.
11. Disposable gloves.
12. Lubricant jelly.
13. Tuning fork (128 Hz and 256 Hz).
14. Two tubes (filled with tobacco and soap).
15. Three papers (for history, speech examination, and tremor examination).
16. Pulse oximetry (sometimes required).

Postgraduate medical candidates need an ophthalmoscope in addition to the above mentioned equipments. No excuse is accepted if the candidates miss any one of them.

4 RAJOOJ'S CLINICAL SKILLS

MEDICAL HISTORY

The history will vary according to whether it is being taken in the wards, the out - patient clinic, the emergency unit or the home and also according to the availability of time and status of the patient. In hospital where teaching is ward – based, the history consists of:

1. PERSONAL HISTORY

BOX 1 – 1. Elements of personal history

M; Marital status
A; Age
R; Religion
A; Address
D; Date of admission
O; Occupation
N; Name and Next of kin
A; ABO and RH group

A useful mnemonic to the elements of personal history recalls MARADONA, a famous footballer

From the medicolegal point of view, every patient admitted into the hospital should have an identity record (BOX 1 – 1) that is unique from other parts of history in that it is a stereotyped routine of asking a standard questions and recording the answers.

Name of the patient should be recorded as it may give a clue to many issues including religious aspect where Muslims tend to nominate Mohammed, Ali or Omer etc... On the other hand, Christ faithful nominates Tom, Hennery or Josef. Other issue is the social aspect where this may reflect the social status of the patient. In addition, a name may be an indication for a previous event happened at the time of birth.

Address reflects the environment in which the patient is living and liability to acquire endemic diseases. Address may reflect the socioeconomic state of the patient. Certain age groups are liable to special diseases; minimal change disease accounts for nephrotic syndrome in most children, dementia usually affects old patient while inflammatory bowel diseases and lymphoma affect middle aged patient as a first peak onset. Occupation of the patient may give a hint at the underlying diseases for example diffuse parenchymal lung disease and lung cancer has been seriously linked to the job. Next of kin identification is important to inform of the history instead a critically ill patient and to receive the instructions about the patient's health state.

Marital status of the patient is inquired as some infectious diseases are sexually transmitted to the partner e.g. gonorrhea and hepatitis B. Religion is also vital as Moslems and Jews for example are almost always immune from penile carcinoma due to circumcision. Ashkenazi Jews are more liable to inflammatory bowel diseases. ABO and RHESUS groups are inquired whenever an inadequate circulation is thought to be the presenting complaint or as a preparation for surgical operation. Date of admission (DOA) should be recorded to identify the progression in the patient's health.

Certain examiners dislike the routine asking of religion and blood group. It is advisable to routinely asking about them and not to present them unless the case is related e.g. in case of bleeding or pallor the blood group should be notified and in an area where more than one religion is present the candidate should ask about the religious status of the patients.

When undergraduate or postgraduate students want to present their case it is sometimes preferable to follow the guidelines provided by their senior lecturers at the time of clinical training course or to follow the examples provided

EXAMPLES

Mr. (Mister) Mohamed Thamer Rahman Altai is seventy years old, living in Diwaniya/ Alaskan quarter. He is married and had three daughters and two sons. He is a teacher. His next of kin is Ali Hussein Rahman Altai; his cousin. The patient is admitted into the hospital on sixteenth of January 2009.

Mr. (Mister) Muhsen Yaseen Kadhem Alali is fifty five years old, living in Najaf/ Alnaser quarter, and is unmarried. He is a manual worker and his blood group is A positive. His next of kin is Ali Muhsen Yaseen Altai; his son. The patient is admitted into the hospital on 7th of March 2009.

Ms (Miss) Maha Majeed Ali Alsalami is thirty years old, living in Baghdad/ Alamel quarter. She is Moslem and unmarried. She is a teacher. Her next of kin is Salma Majeed Ali Alsalami; her sister. The patient is admitted into the hospital on seventh of September 2011.

Mrs. (Mistress) Nora Josef Tomas is forty years old, living in Basra/ Alashar quarter. She is Christian, married and had one daughter and two sons. She is a house wife. Her next of kin is Yara Josef Tomas; her sister. The patient is admitted into the hospital on sixteenth of February 2010.

2. CHIEF COMPLAINT (S) AND ITS DURATION

A brief statement of the main problem (s) that brought the patient to the hospital and the duration of the complaint (BOX 1 – 2). In fact, this statement is the patient's own wording and not medically correct terms.

BOX 1 – 2. Examples of the chief complaint

1. Shortness of breath for three days prior to admission.
2. Abdominal pain for eight hours prior to admission.
3. Coughing up blood for three hours prior to admission.
4. Vomiting up of blood for one day before admission.
5. Passing of black tarry stools for ten days prior to admission.
6. Yellowish discoloration of the skin, mucous membrane and sclera for two days prior to admission.

Note the wording. Use the patient's own language and not medically correct terms like dyspnoea, acute abdomen, hemoptysis, hematemesis, melena, or jaundice.

A patient presented with multiple complaints e.g. SOB for ten days prior to the time of admission, cough for seven days, and chest pain for five days, thereafter the patient started to cough up of blood for one day and the admission was advised. Thus the chief complaint is coughing up blood for one day prior to admission.
The following topics as illustrated in BOX 1 – 3 seem appropriate for Iraqi patients (non – English speakers).

BOX 1 – 3. Patient's descriptions of medical terms

Medical terms	Patients description	Medical terms	Patients description
Headache	Headache	Diarrhea	Frequent bowel motions
Fever	Fever	Constipation	Inability to pass stool
Coma	Loss of consciousness	Obstipation	Complete constipation with no passage of either feces or gas
Terminal dribbling	Difficulty in stopping micturition	Jaundice	Yellowish discoloration of the skin, mucous membrane and sclera
Dyspnea	Shortness of breath (SOB)	Melena	Black tarry stool
Palpitation	Awareness of heart beats	Loin pain	Loin pain
Chest pain	Chest pain	Frequency	Frequent micturition
Edema	Ankle or feet swelling	Urgency	Rapid desire for micturition
Cough	Cough	Dysuria	Painful micturition
Vomiting	Vomiting	Edema of face	Puffiness of the face
Abdominal pain	Abdominal pain	Polydipsia	Increased water intake
Hemoptysis	Coughing up blood	Odynophagia	Painful swallowing
Cyanosis	Bluish discoloration of the body	Dysphagia	Difficulty in swallowing
Retention	Inability to pass urine	Thyrotoxicosis	Easy fatigueability
Lump	A swelling e.g. in the abdomen	Fatigue	Generalized weakness of the body
Anemia	Pallor of the skin	Ascites	Abdominal distension
Arthritis	Limitation of joint movement	Tenesmus	Feeling of incomplete defecation
Hematemesis	Vomiting up blood	Hematochezia	Bleeding per rectum or rectal bleeding
Dysgeusia	Altered taste sensation	Water brash	Excessive salivation
Globus	Feeling of a lump in the throat	Polyphagia	Increased food intake
Intermittent claudication	Pain in calf on walking	Indigestion/ Dyspepsia	Discomfort after eating
Vertigo	Sensation of unsteadiness	Dizziness	Sensation of faintness on standing
Hesitancy	Difficulty in initiating micturition	Regurgitation	Return of food into the mouth
Anorexia	Loss of appetite	Pneumaturia	Passing of gas bubbles with the urine
Cacageusia	Foul taste in mouth	Gynecomastia	Breast enlargement in male
Heart burn	Burning sensation in the chest	Paresthesia	Tingling sensation (numbness)
Halitosis	Bad breath	Xerostomia	Dry mouth
Hiccups	Hiccups or hiccoughs	Convulsion	Jerking movement of hands/face
Geophagia	Desire to ingest clay	Hyperhidrosis	Excessive sweating
Pagophagia	Desire to ingest ice	Anismus	Difficulty emptying the rectum

3. HISTORY OF PRESENT ILLNESS

The public is increasingly informed on the daily clinical sessions in the wards, but they still seek medical help from white coated persons. In addition, doctors and medical students retain a respected status in the society. This appreciation will help you in establishment or renewal a successful patient-doctor relationship. The corner stone of such a relationship is to establish the heart of the history when you gently ask the patient (tell me about your complaint (s), in details and from the beginning). When the patients talk about their trouble, they will expect a high standard of respect and security, therefore, a strict ethical behavior on the part of the doctor is required in order to set the scene for a trusting relationship. Remember that tactful encouragement is always necessary to get the full details of the history and to avoid unnecessarily leading questions which the patient may not understand. Over talkative patients who insist to runaway from the point of discussion and to use pseudomedical terms without actually understanding the medical meaning should be kindly discouraged and asked to describe what they actually feel to be wrong. For whom in addition, an attempt should always be made to indicate particularly important axes of the questions.

Many patients find considerable difficulty putting their symptoms into words particularly when their symptoms are outside ordinary experience. For those patients it may be helpful to supply a list of alternatives for the presenting complaint e.g. where is the site of pain; is it deep, inside? Then to correlate the relevant answer into suitable meaning.

Other patients may have multiple complaints, particularly old patients that are impossible to identify a single systemic involvement. In these instances, try to find out the actually system (s) involved, and to analyze its symptoms while other semi-involved system (s) should be deferred into the next step of history where leading question is of value.

Once you complete a single chunk of the history, you have to ask (what happened next?) this will provide a fertile ground for the elements of the analysis. It is necessary to record all the details of the history in every patient because at the start it is difficult to separate the important from the trivial. In addition some negative findings may be relevant.

With increasing experience, it becomes easier to judge what does and does not need to be omitted safely.

The details of the history must not be divulged to any one who is not from the medical staff, but must be shared in concise form to other students or doctors. The students should practice oral presentation of their cases to the senior lecturer at every opportunity in temporal and coherent order making the history an interesting and easily grasped story. A genuine interest in the case presented combined with gentleness of manner allow both a student and his/her senior lecturer to feel at ease while the process of presentation continues.

BOX 1 – 4. Guidelines for the history of present illness

Analysis of the chief complaint (s) in the form of patient's own wording.
Analysis of the associated symptom (s).
Asking about the other symptoms of the involved system.
Basic investigation that the patient had undergone meanwhile admission.
Progression in the health status of the patient in the form of improvement or worsening since date of admission up to the time being now.
In case of surgical history, mention the patient's condition post operatively in the form of fluid intake, medications, passing of flatus and movement of the patient.

Finally the history of present illness should be summarized as a story and reiterated for the patient to confirm and correct. Generally speaking, the history of present illness should follow the guidelines provided in the medical school or the students follow the points in BOX 1 – 4.

CLINICAL CLUE

A 25 – year – old women presented chiefly due to abdominal pain that is associated with vomiting. How can you analyze her complain? Abdominal pain is the chief complain that should be thoroughly analyzed and the associated symptom is vomiting that should also be thoroughly analyzed. Other symptoms e.g. diarrhoea or constipation of the involved system (GIT) should also be inquired.

PRESYNCOPE AND SYNCOPE

Presyncope is the feeling of lightheadedness and near-collapse while syncope (faint) is defined as a transient brief sudden loss of consciousness due to global cerebral hypoperfusion with rapid onset, short duration and spontaneous recovery; vision often darkens and there may be a ringing in the ears. The European Society of Cardiology published in 2009 suggested the following classification of syncope:

I. Reflex syncope is neurally mediated and includes
- Vasovagal: triggered by emotion, pain, or stress.
- Situational syncope: cough, micturition, gastrointestinal.
- Carotid sinus syncope.

II. Orthostatic syncope
- Primary autonomic failure: Parkinson's disease, Lewy body dementia.

- Secondary autonomic failure: diabetic neuropathy, amyloidosis, and uremia.
- Drug – induced: diuretics, alcohol, vasodilators.
- Volume depletion: hemorrhage, diarrhea.

III. Cardiac syncope

- Arrhythmias: bradycardia (sinus node dysfunction, atrioventricular conduction disorders) or tachycardia (supraventricular and ventricular).
- Structural: valvular, myocardial infarction, hypertrophic obstructive cardiomyopathy.
- Others: pulmonary embolism.

On the other hand coma is defined as a persistent loss of consciousness due to disorder of the arousal mechanisms in the brain stem and diencephalon and indicates bilateral hemisphere or brain-stem disease.

CLINICAL CLUE

Persistent loss of consciousness means coma while transient episodic loss of consciousness indicates syncope or seizure (fits)

As a transient phenomenon, loss of consciousness most commonly occurs because of syncope or alternatively loss of consciousness occurs from sudden electrical dysfunction of the brain during a seizure (fit). Episodes of loss of consciousness are therefore either fits or faints, though many patients who have various types of psychogenic blackout confuse this clear distinction. The distinction of a seizure from a faint can only be made from the patient's history, with help from a witness to the attack. Features in the history useful in distinguishing a seizure from a faint are shown in BOX 1 – 5.

BOX 1 – 5. Features helpful in distinguishing seizure from syncope (faint)

	Seizure	Faint
Aura (e.g. olfactory)	+	-
Cyanosis	+	-
Tongue-biting	+	-
Post-ictal confusion	+	-
Post-ictal amnesia	+	-
Post-ictal headache	+	-
Rapid recovery	-	+

CLINICAL CLUE

It is rare for the syncope to cause injury and there is no amnesia for events that occur after regaining awareness. During a syncopal attack, incontinence of urine can occur and there is often stiffening and brief twitching of the limbs, but tongue-biting never occurs.

PRESENTATION OF SYNCOPE: TRANSIENT LOSS OF CONSCIOUSNESS

1. What are the symptoms preceding syncope? In cardiac syncope, there is often none but there may be lightheadedness, palpitation, chest pain, or breathlessness while in vasovagal syncope, there is often nausea, lightheadedness, or sweating.
2. What is the behavior of the patient during the syncope? In cardiac syncope, the patient looks extremely dead-like' while in vasovagal syncope, there is often striking pallor.
3. What are the associated symptoms during the syncope? During a syncopal attack, incontinence of urine can occur and there is often stiffening and brief twitching of the limbs, but tongue-biting never occurs.
4. What is the duration of the syncope? In cardiac syncope, the patient rapidly recovers in less than one minute with flushing while in vasovagal syncope, the recovery is often slow and there are nausea and lightheadedness.
5. What are the symptoms that follow the syncope? It is rare for the syncope to cause injury and there is no amnesia for events that occur after regaining awareness.
6. What are the provoking factors? Drugs, alcohol, pain, stress, cough, or micturition or the patient is a known case of cardiac problems.

EXAMPLES

The condition started suddenly as a transient loss of consciousness whilst the patient was sitting. The attack generally lasted less than one minute during which the patient became dead-like and mildly stiff when the patient's son tried to shake him. The condition was also associated with brief twitching of both upper and lower limbs, no incontinence of urine, and no tongue biting. The patient rapidly recovered in less than one minute; feeling slightly lightheaded and having no nausea or headache. He immediately was able to retain the events prior to this attack. He experienced that he had not consumed alcohol or used any medicine prior to this attack. In addition, he had no SOB or awareness of heart beats but chest pain that was.....

What is the diagnosis?
Such a presentation should raise suspicion of cardiac syncope e.g. aortic stenosis.
If such an attack occurring in young adults during or after physical exertion and resulting in death, what do you expect the cause?
The cause is probably hypertrophic cardiomyopathy.

The condition started suddenly as a transient loss of consciousness whilst the medical student was standing looking at pleural aspiration procedure. The attack generally lasted less than one minute during which the student became pale, sweaty, and mildly stiff when his friends rushed to awake him. The condition was also associated with brief twitching of both upper and lower limbs, no incontinence of urine, and no tongue biting. The patient slowly recovered in 5 minutes; feeling slight lightheadedness and nausea. After that, he was able to retain the events prior to this attack. He experienced that he had not consumed alcohol or used any medicine but felt lightheadedness and nausea prior to the attack.

What is the diagnosis?
Such a presentation should raise suspicion of vasovagal syncope.

DIZZINESS AND VERTIGO

Dizziness is an umbrella term describing symptoms varying from vague lightheadedness ("I'm just dizzy" that is usually due to anxiety or "I'm going to faint" i.e. presyncope) to disorientation, disequilibrium ("my balance is off" or "I feel as if I'm going to fall") or vertigo. Vertigo means sensation of unsteadiness and the illusion of movement of either the body or the surroundings, typically spinning or rotating. BOX 1 – 6 shows the common causes of vertigo.

BOX 1 – 6. Common causes of vertigo

Central	Peripheral
Vertebrobasilar insufficiency	Ménière's disease
Brainstem or cerebellum ischemia or infarction	Benign paroxysmal positional vertigo (BPPV)
Multiple sclerosis	Vestibular neuronitis (acute labyrinthitis)
Migraine	Drugs, e.g. gentamicin, anticonvulsants
	Cerebellopontine angle tumors e.g. acoustic neuroma

CLINICAL CLUE

The peripheral localization of vertigo is suspected when the attack is marked, having finite duration (minutes, days, weeks) but recurrent, alone or in combination with nausea and vomiting, with or without deafness and tinnitus, and no neurologic findings.
The central localization of vertigo is suspected when the attack is mild in severity, having protracted course, rarely alone, and is confirmed by neurologic findings of involvement of the brainstem (cranial nerves, sensory and motor tracts, etc.) or cerebellum (cerebellar ataxia with eyes open, nystagmus, dysarthria, etc.) that accompanied the attack as well as normal hearing function (no deafness or tinnitus).

Benign paroxysmal positional vertigo is by far the most common type of vertigo. Patients with this condition suddenly develop brief episodes of vertigo (few seconds to minutes) with position change, typically when turning over in bed, getting in and out of bed, bending over and straightening up, or extending the neck to look up. It is sometimes associated with nausea and no hearing loss, no deafness, and no neurologic symptoms. The course of vertigo is persistent for few weeks and recurrent. It commonly occurs spontaneously in older people. Benign positional vertigo results when otolith debris inadvertently enters one of the semicircular canals. Vestibular neuronitis (acute labyrinthitis) describes the sudden onset of vertigo, nausea, and vomiting lasting hours to days, up to two weeks and not associated with deafness, tinnitus, or neurologic symptoms. Most affected patients gradually improve over 1 to 2 weeks although vertigo may recur over 12 – 18 months. Many report an upper respiratory tract illness 1 to 2 weeks before the onset of vertigo. Ménière's syndrome is a condition associated with fluctuating hearing loss and tinnitus, recurrent episodes of abrupt and often severe vertigo lasting several hours to a day or more, a sensation of fullness or pressure in the affected ear, and nausea and vomiting. Drugs toxicity may cause acute or insidious onset of vertigo that may or may not be reversible, associated with nausea and vomiting, and may be associated with hearing loss in both sides and tinnitus. Most cerebellopontine angle tumors (acoustic neuroma, meningioma, epidermal cyst) grow slowly, allowing the vestibular system to accommodate so that they produce only a vague sensation of disequilibrium or insidious onset of vertigo that has variable course and duration and associated with symptoms of pressure on cranial nerves V, VII, and VIII. In virtually all patients, hearing loss in one side and tinnitus is present.
Vertebrobasilar insufficiency is usually caused by atherosclerosis of the subclavian, vertebral, and basilar arteries and is a common cause of vertigo in elderly persons. Vertigo with vertebrobasilar insufficiency is abrupt in onset, usually lasting several minutes, and is frequently associated with nausea and vomiting. Associated symptoms resulting from ischemia to the posterior circulation include visual illusions and hallucinations, drop attacks and weakness, visual field defects, diplopia, and headache. These symptoms occur in episodes either in combination with the vertigo or alone. Vertigo is a common symptom with infarction of the lateral brain stem or cerebellum, or both. The diagnosis is usually clear, based on the characteristic acute history and neurologic findings. Vertigo as the sole manifestation of brainstem disease is rare unless other symptoms and signs of brainstem disorder appear within several days (cranial nerves, sensory and motor tracts, etc.). The same is true of multiple sclerosis, which may be the explanation of a persistent vertigo in an adolescent or young adult. Vertigo of cerebellar origin is exceptional in this respect in that it may rarely be the sole manifestation of cerebellar infarction or hemorrhage. Vertigo in multiple sclerosis is usually transient and often associated with other neurologic signs of brain stem disease, in particular, internuclear ophthalmoplegia or cerebellar dysfunction. Vertigo is a common symptom with migraine. It can occur with headaches or in separate isolated episodes, and it can predate the onset of headache.

HOW TO APPROACH TO DIZZINESS?

SOCRATES

S: Subjective or objective O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Duration and Course) E: Exacerbating and precipitating factors S: Severity

PRESENTATION OF DIZZINESS

1. What did the patients mean by dizziness? Do they feel just dizzy? Do you feel unsteady, as if you are going to fall or black out? Or do they feel the room is spinning (true vertigo)? In true vertigo, there is actually a sense of motion, usually of the surroundings but also of the head itself. When vertigo is severe it may not be possible for patient to stand or walk. Some patients may be able to identify their symptoms only when asked to compare them with the feeling of movement they experience when they come to a halt after rapid rotation.
2. What is the mode of onset of dizziness e.g. sudden or gradual?
3. What is the character of vertigo (Central or peripheral)?
4. What are the relieving factors e.g. sleep or medications?
5. What is the timing of vertigo (duration and course)?
6. What are the associated symptoms e.g. nausea, vomiting, headache, hearing loss, ringing in the ears (tinnitus), or neurologic findings?
7. What are the triggering or exacerbating factors for vertigo e.g. change in position, drugs, or trauma?
8. What is the severity of vertigo?

WHERE IS THE LESION SITE IF A PATIENT PRESENTED WITH VERTIGO AND:

1. Nausea, vomiting, and deafness?
2. Diplopia and drop attacks?
3. Ipsilateral facial weakness and limb ataxia?

ANSWER IS

1. Peripheral (labyrinthine) lesion.
2. Central (posterior cerebral circulation) lesion.
3. Cerebellopontine angle.

EXAMPLES

The condition recurred on awakening in this morning as a feeling of unsteadiness and motion of the head and the surroundings. Four previous attacks occurred during the previous two days. Each attack lasted for about 1 minute and was moderately severe rendering the patient disliking to change his head position as it was mainly triggered by such an act. There was no associated tinnitus, deafness, hiccups or double vision; just a sense of nausea that is followed by vomiting and headache.

What is the diagnosis?
The diagnosis is peripheral (labyrinthine) vertigo e.g. BPPV.

The condition started on awakening this morning as a feeling of unsteadiness and motion of the head and the surroundings. The attack was mild, persisting for more than several minutes and was associated with double vision, drop attacks and weakness, nausea and vomiting. There was no associated tinnitus or deafness. There was no prior history of drugs intake

What is the diagnosis?
The diagnosis is central vertigo due to vertebrobasilar insufficiency.

HEADACHE AND FACIAL PAIN

Headache results from disorders that affect pain-sensitive structures of the head and neck. Relatively few cranial structures are pain-producing; these include the scalp, middle meningeal artery, dural sinuses, and falx cerebri. Headaches that are new in onset or different from previous headaches are those most likely to be caused by a serious illness, whereas headaches of long standing usually have a benign cause.

CLINICAL CLUE

Acute, severe headache with stiff neck and fever suggests meningitis. Acute, severe headache with stiff neck but without fever suggests subarachnoid hemorrhage.

HOW TO APPROACH TO HEADACHE?

S: Site O: Onset C: Character R: Radiation A: Associated factors
T: Timing (Duration and Course) E: Exacerbating, precipitating, and relieving factors S: Severity

SOCRATES

PRESENTATION OF HEADACHE: HEADACHE

1. Where is the site of headache? Unilateral headache is an invariable feature of cluster headache and the majority of migraine attacks; most patients with tension headache report bilateral pain. Ocular or retroocular pain suggests a primary ophthalmologic disorder, migraine or cluster headache (FIGURE 1 – 1). Paranasal pain localized to one or several of the sinuses, often associated with tenderness in the overlying periosteum and skin, occurs with acute infection (acute sinusitis). Headache from intracranial mass lesions may be focal ("it hurts right here"), but even in such cases it is replaced by bioccipital and bifrontal pain when the intracranial pressure becomes elevated. Occipital discomfort is commonly associated with tension headache. Occipital localization can also occur with meningeal irritation from infection or hemorrhage and with disorders of the joints, muscles, or ligaments of the upper cervical spine. Pain within the first division of the trigeminal nerve is a common feature of post – herpetic neuralgia. Lancinating pain localized to the second or third division of the trigeminal nerve suggests trigeminal neuralgia (tic douloureux).

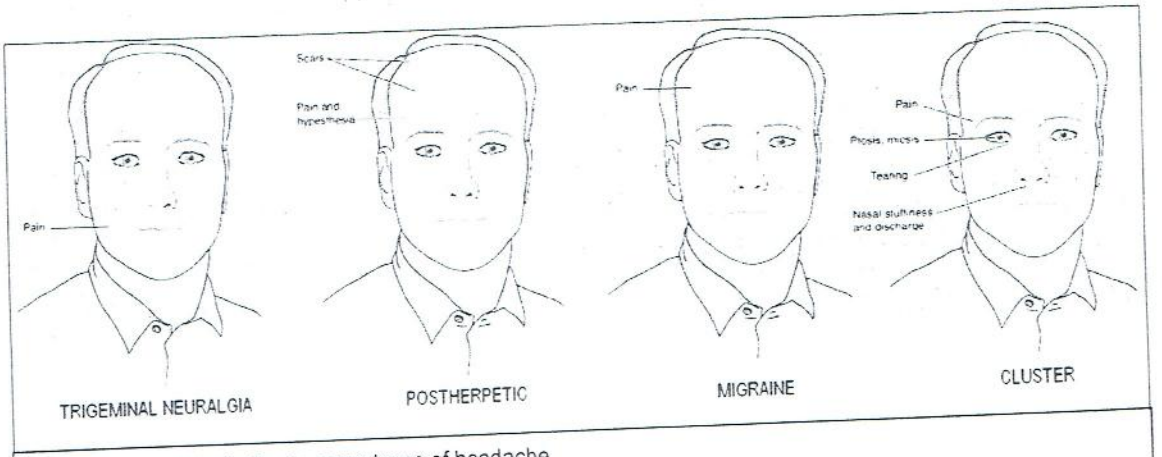


FIGURE 1 – 1. Distribution in some types of headache.

2. What is the onset of the pain? BOX 1 – 7 shows some causes of headache and facial pain according to the onset.

BOX 1 – 7. Causes of headache and facial pain according to the onset

Acute onset	Subacute onset	Chronic onset
Meningitis or encephalitis	Giant cell (temporal) arteritis	Migraine
Subarachnoid hemorrhage	Intracranial mass	Cluster headache
Other cerebrovascular diseases	Pseudotumor cerebri	Tension headache
Ocular disorders (glaucoma, acute iritis)	Trigeminal neuralgia	Cervical spine disease
Seizures	Posttherpetic neuralgia	Sinusitis and dental disease

3. What is the character of headache? Pulsating, throbbing pain is frequently ascribed to migraine, but it is equally common in tension headache. A steady sensation of tightness or band-like or pressure is also commonly seen with tension headache. The pain produced by intracranial mass lesions is typically dull and steady. Sharp, lancinating pain suggests a neuritic cause such as trigeminal neuralgia.
4. What is the radiation of headache? Tension headache may arise from the occipital region to involve the whole areas in the head.
5. What are the associated symptoms? Recent weight loss may accompany cancer or giant cell arteritis. Fever or chills may indicate systemic infection or meningitis. Visual disturbances suggest an ocular disorder, migraine, or an intracranial process involving the optic nerve or tract or the central visual pathways. Nausea and vomiting are common in migraine and can be seen in the course of mass lesions. Some patients with migraine also report that diarrhea accompanies the attacks. Photophobia may be prominent in migraine and acute meningitis or subarachnoid hemorrhage. Myalgias often accompany tension headaches, viral syndromes, and giant cell arteritis. Ipsilateral rhinorrhea and lacrimation during attacks typify cluster headache.
6. What is the timing of headache? Headaches from mass lesions are commonly maximal on awakening, as are sinus headaches. Cluster headaches frequently awaken patients from sleep; they often recur at the same time each day or night. Tension headaches can develop whenever stressful situations occur and are often maximal at the end of a workday. Migraine headaches are episodic and may last 4 – 72 hours. In contrast to migraine, tension headache may continue for weeks or months without interruption. Cluster headache is characteristically brief and lasting 30-90 minutes.
7. What are the precipitating and exacerbating factors of headache? In some migraineur patients there are identifiable dietary precipitants e.g., cheese, chocolate or red wine. Use of oral contraceptive agents or nitrates may precipitate migraine. Precipitation of headache by alcohol is typical of cluster headache. Chewing and eating commonly trigger tic douloureux, and the jaw claudication of giant cell arteritis. Discomfort by rapid changes in head position or by events that transiently raise intracranial pressure, such as coughing and sneezing, is often associated with an intracranial mass (BOX 1 – 8) but can occur in migraine. Anger, excitement, or irritation can precipitate or worsen both migraine and tension headaches. Stooping, bending forward, sneezing, or blowing the nose characteristically worsens the pain of sinusitis. Postural headache (maximal when upright, nearly absent when lying down) occurs with low cerebrospinal fluid (CSF) pressure caused by lumbar puncture, head injury, or spontaneous spinal fluid leak. Fluctuations in intensity and duration of the headache with no obvious cause, especially when associated with similar fluctuations in mental status, are seen with subdural hematoma.
8. What are the factors relieving headache? Migraine are frequently relieved by darkness, sleep, vomiting, or pressing on the ipsilateral temporal artery, and their frequency is often diminished during pregnancy. Postlumbar-puncture and low-pressure headaches are typically relieved by recumbency, whereas headaches caused by intracranial mass may be less severe with standing.

9. What is the severity of headache? The severity of headache is assessed as to whether or not the headache interferes with sleeping, eating, talking, walking, or other usual daily activities.

CLINICAL CLUE

Diagnostic criteria for migraine headache require repeated attacks of headache lasting 4–72 h in patients with a normal examination, no other reasonable cause for the headache, and:
At least two of the following features:

1. Unilateral pain
2. Throbbing pain
3. Aggravation by movement
4. Moderate or severe intensity

Plus at least 1 of the following features:

1. Nausea/vomiting
2. Photophobia and phonophobia.

DRAW 3

BOX 1 – 8. Headache of raised intracranial pressure

Dull ache, often mild	Worse in morning, improves through the day
Relieved by analgesia	Worse bending forward
Associated with morning vomiting	Worse with cough and straining

CLINICAL CLUE

Most migraine aura symptoms develop over 5 to 20 minutes and usually last less than 60 minutes. The aura can be characterized by visual, sensory, or motor phenomena. Headache, when present, usually occurs within 60 minutes of the end of the aura. The most frequently occurring aura is visual in nature e.g. scotomata, simple flashes, shimmering of light or silvery zigzag lines. Paresthesias are the second most common aura: a spreading front of tingling followed by numbness which moves, over 20-30 minutes, from one part of the body to another.

It is best to look upon migraine as a triad of paroxysmal headache, nausea and/or vomiting, and an 'aura' of focal neurological events (usually visual). Patients with all three of these features are said to have classical migraine. Those with paroxysmal headache (with or without vomiting) but no 'aura' are said to have common migraine. In some patients the focal neurological events (auras) may occur by themselves ('migraine equivalent') without headache, but in this case other structural disorders of the brain, or even focal epilepsy, need to be considered in the differential diagnosis. In a smaller number of patients, the symptoms of the aura do not resolve, leaving more permanent neurological disturbance ('complicated migraine').

EXAMPLES

The condition started almost every day for the preceding three years as headache that initially localised to the back of the head then became generalized to involve the whole head, mild in intensity, and band- like in nature, although at certain occasions became throbbing. The patient experienced headache almost every day particularly in the evening and became less noticeable in the morning. This headache was not associated with nausea or vomiting, or visual disturbance apart from very infrequent occasion of muscle ache, exacerbated during stress and relieved when the patient was busy or went to bed.

What is the diagnosis?
The diagnosis is tension headache.

The condition started as visual disturbance in the form of zigzag lines marching across the right eye over 20 minutes then she developed headache one hour after the end of the initial visual complain. She experienced headache once monthly for the previous three years that was localised the right side of the head and face, mild in intensity at the start then became of moderate severity, and pulsating in nature. The headache precipitated with ingestion of chocolate and persisted for 24 hours. During this time, the patient had nausea and vomiting and preferred to sleep and stay in dark environment. The patient had an exacerbation during menses and slight improvement when became pregnant or developed vomiting.

What is the diagnosis?
The diagnosis is migraine headache.

The condition started as headache and right eye pain that were awaken him from sleep in the early hours of the day and exactly recurred in the same time each day for around 4 weeks before it disappeared but after 3 months the headache and facial pain recurred again. The headache was localised to the right side of the head and face, moderately severe in intensity, dull in nature, and relieved with oxygen therapy. The headache precipitated by alcohol intake and lasted for 30 minutes only. During this time, the patient had right nasal congestion and right eye tearing.

What is the diagnosis?
The diagnosis is cluster headache.

The condition started every day in the morning for the preceding ten days as headache that was involving the whole front and back of the head. The headache was mild in intensity, dull ache in character, associated with early morning vomiting and visual disturbance, and slightly improved through out the day. The headache was exacerbated with cough, straining, and bending forward and improved pain medication.

What is the diagnosis?
The diagnosis is headache of raised intracranial pressure

SEIZURES: EPILEPSY

A seizure is a paroxysmal event due to abnormal, excessive, hypersynchronous discharges from an aggregate of central nervous system (CNS) neurons that may be manifested as an impairment or loss of consciousness, abnormal motor activity, sensory disturbance, autonomic dysfunction as well as behavioral abnormalities. The meaning of term seizure needs to be carefully distinguished from that of epilepsy. Epilepsy describes a condition in which a person has recurrent seizures due to a chronic underlying process. This definition implies that a person with a single seizure, or recurrent seizures due to correctable or avoidable circumstances, does not necessarily have epilepsy. Patients are usually presented with a history of blackout.

CLINICAL CLUE

What is meant by blackout?
Some patients mean by blackout that their vision darkens without alteration in consciousness. More often (blackout) is used to describe an episode of lost consciousness with or without falling down.

The chief division of seizure types is between partial (focal) seizures in which the abnormal activity is limited to one part of the cortex, and generalised seizures where the abnormality involves both hemispheres simultaneously and

synchronously. If partial seizures remain localised, the symptomatology depends on the cortical area affected. If consciousness is preserved, the attack is termed a 'simple partial seizure'. However, if the activity involves parts of the brain concerned with awareness, then consciousness is affected and a 'complex partial seizure' results. If partial seizure further spread into the remainder of the cortex the term a secondary generalised seizure is applied. In primary generalised seizures, the abnormal activity begins synchronously throughout the cortex without an initial partial onset.
Partial (focal) seizures are classified into motor, sensory, autonomic, and psychic seizure.
Partial seizure with motor components is typically clonic i.e. rhythmic muscle contractions and relaxations or jerking movement; pure tonic seizure i.e. increased tone or rigidity of the muscles may be seen as well. Partial seizures with sensory components manifest as changes in somatic sensation (numbness, tingling, or a "pins-and-needles" feeling and occasionally as a sensation of crawling), vision (flashing lights or formed hallucinations), and equilibrium (sensation of falling or vertigo). Partial seizures with autonomic components manifest as changes in autonomic function (flushing, sweating, piloerection). Partial seizures arising from the temporal or frontal cortex cause psychic symptoms. This includes the sensation of unusual intense odors (burning rubber or kerosene) or sounds (crude or highly complex sounds), or an epigastric sensation that rises from the stomach or chest to the head. Some patients describe odd, internal feelings such as fear, feelings of unreality (jamais vu) or undue familiarity (déjà vu) with the surroundings, or illusions that objects are growing smaller (micropsia) or larger (macropsia). These subjective, "internal" events that are not directly observable by someone else are referred to as auras when precede a complex partial or secondarily generalized seizure.

CLINICAL CLUE

What do we mean by Jacksonian march', Todd's palsy, and epilepsia partialis continua?
All these phenomena are features of partial seizure with motor components. Some epileptic activity begins in a very restricted region (e.g. mouth, thumb, great toe etc.) and spread gradually (over seconds to minutes) to include a larger portion of the extremity; this is 'Jacksonian march' and is true for both sensory and motor seizure. Motor attacks vary in duration from a few seconds to several hours. More prolonged episodes may leave paresis of the involved limb lasting for minutes to many hours in the involved region after the seizure ceases (Todd's palsy). In rare instances the partial seizure may continue for hours or days and termed epilepsia partialis continua.

Partial seizures (whether simple or complex) can spread to involve both cerebral hemispheres and produce a generalized seizure, usually of the tonic-clonic variety (secondary generalised tonic – clonic seizure). A partial seizure with secondary generalization is often difficult to distinguish from a primary generalized tonic-clonic seizure, since bystanders tend to emphasize the more dramatic, generalized convulsive phase of the seizure and overlook the more subtle, focal symptoms present at onset.
In complex partial seizure there are usually three phases: the simple partial seizure (described above) or more commonly the auras, the phase of impaired consciousness and behavioral arrest, and the automatism. The aura is usually short- lived; lasting a few seconds or longer. The symptoms in the aura can take any of the forms; epigastric sensations are most common, but affective (fear), psychic (déjà vu), and sensory (olfactory hallucinations) symptoms also occur. The patients retain some memory for the first phase but will have amnesia for the remaining phases. In the behavioral arrest or motionless stare phase, both motor and speech are arrested. Behavioral arrest is usually accompanied or immediately followed by automatisms, which are involuntary, automatic behaviors that consist of very basic behaviors such as chewing, lip smacking, swallowing, or "picking" movements of the hands, or more elaborate behaviors such as a display of emotion or running. The patient is typically confused following the seizure, and the transition to full recovery of consciousness may range from seconds up to an hour.

CLINICAL CLUE

What is meant by aura?
The aura is the portion of the seizure that precedes loss of consciousness in complex partial seizure and partial seizure with secondary generalization and of which the patient retains some memory. It is reserved for subjective internal symptoms of epileptic origin reported by the patient in the absence of objective signs. The aura is sometimes the sole manifestation of the epileptic discharge.

Classification of seizure types and causes of seizure are shown in BOX 1 – 9.

BOX 1 – 9. Classification and causes of seizure

CLASSIFICATION OF SEIZURE	
Focal (partial) seizure	Generalized seizure
Simple partial seizure Complex partial seizure Partial seizure with secondary generalization	Tonic clonic (grand mal epilepsy) Tonic seizure Clonic seizure Atonic seizure Myoclonic seizure Absence seizure (petit mal epilepsy)

CAUSES OF SEIZURES	
Idiopathic	Some types of the benign epilepsies of the childhood
Genetic	Tuberous sclerosis, neurofibromatosis, and von Hippel-Lindau disease
Developmental	Hydrocephalus
Tumours	Primary and secondary brain tumors
Trauma	Including surgery
Vascular	Cerebrovascular accident (CVA)
Infections	Meningitis, encephalitis, cerebral abscess, and toxoplasmosis
Inflammation	Vasculitis (SLE) and multiple sclerosis (uncommon)
Metabolic	Hypocalcemia, hyponatremia, hypomagnesemia, and hypoglycemia Renal failure and liver failure
Drugs, alcohol and toxins	Antibiotics: penicillin, isoniazid, metronidazole Psychotropic agents: phenothiazines, tricyclic antidepressants, lithium
Degenerative	Alzheimer's disease (uncommonly)

Primary generalized tonic-clonic seizures are attacks in which consciousness is lost and usually without warning. When a warning does occur, it usually consists of nonspecific vague symptoms e.g. headache, mood change, anxiety, irritability, changes in appetite, or dizziness. This prodrome is distinct from the auras associated with focal seizures that secondarily generalize. The initial manifestations are unconsciousness and tonic contractions (Figure 1 – 2) of muscles throughout the body for 10-30 seconds, producing first flexion and then extension particularly of the back and neck. Tonic contraction of the muscles of respiration may produce an expiration-induced vocalization (ictal cry or moan). Respirations are impaired, secretions pool in the oropharynx, and cyanosis develops. Contraction of the jaw muscles may cause biting of the tongue. The patient falls to the ground and may be injured. The tonic phase is followed by a clonic (alternating muscle contraction and relaxation) phase (Figure 1 – 2) of symmetric limb jerking that persists for an additional 30-60 seconds, or longer. With time, the jerking becomes less frequent, until finally all movements cease and the muscles are flaccid. The postictal phase is characterized by unresponsiveness, muscular flaccidity, and excessive salivation that can cause stridor and partial airway obstruction. Bladder or bowel incontinence may occur at this point. Patients gradually regain consciousness over minutes to hours, and during this transition there is typically a period of postictal confusion. Patients subsequently complain of headache, fatigue, and muscle ache that can last for many hours. The duration of impaired consciousness in the postictal phase can be extremely long, i.e., many hours, in patients with prolonged seizures or underlying CNS diseases such as alcoholic cerebral atrophy. There are many variants of the generalized tonic-clonic seizure, including pure tonic, pure clonic, atonic, and myoclonic seizures.

CLINICAL CLUE

How to differentiate psychogenic non-epileptic attacks ('pseudo-seizures') from seizure?
Clues pointing towards non-epileptic attacks include elaborate arching of the back in an attack, pelvic thrusting and/or wild flailing of limbs. These attacks are not usually followed by the same degree of post-ictal confusion and do not cause cyanosis or tongue biting, but urinary incontinence can occur.

Tonic seizures are characterized by continuing muscle contraction that can lead to fixation of the limbs and axial musculature in flexion or extension and are a cause of drop attacks; the accompanying arrest of ventilatory movements leads to cyanosis. Consciousness is lost, and there is no clonic phase to these seizures. Clonic seizures are characterized by repetitive clonic jerking accompanied by loss of consciousness. There is no initial tonic component. Atonic seizures are characterized by sudden loss of postural muscle tone lasting 1–2 s. Consciousness is

briefly impaired, but there is usually no postictal confusion. A very brief seizure may cause only a quick head drop or nodding movement, while a longer seizure will cause the patient to collapse. This can be extremely dangerous, since there is a substantial risk of direct head injury with the fall. Myoclonus is a sudden, brief, shock – like muscle contractions that may involve one part of the body or the entire body. A normal, common physiologic form of myoclonus is the sudden jerking movement observed while falling asleep.

Typical absence seizures (petit mal seizures) are characterized by sudden, brief lapses of consciousness without loss of posture. The seizure typically lasts for only seconds, consciousness returns as suddenly as it was lost, and there is no postictal confusion. Although the brief loss of consciousness may be clinically inapparent, absence seizures are usually accompanied by subtle, bilateral motor signs such as rapid blinking of the eyelids, chewing movements, or small-amplitude, clonic movements of the hands.

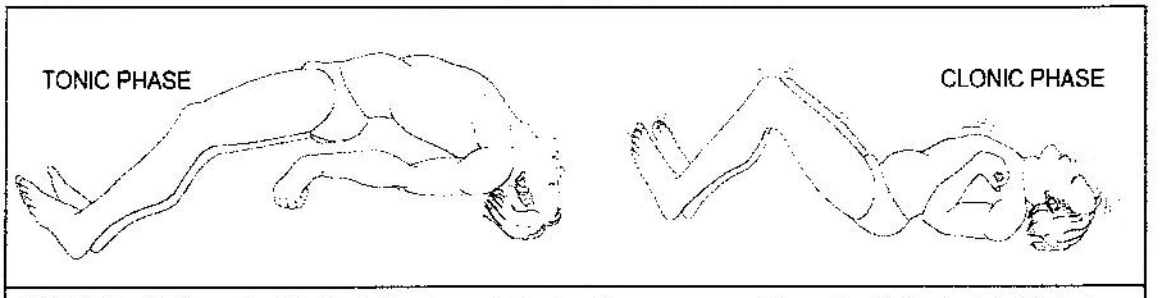


FIGURE 1 – 2. Generalized tonic-clonic seizure, illustrating the appearance of the patient in the tonic (stiffening) and clonic (shaking) phases.

PRESENTATION OF SEIZURES: JERKING MOVEMENT OF LIMBS OR RIGHT SIDE...ETC

1. What are the events that preceded the convulsion e.g. the aura or simple partial seizure before secondary generalised tonic – clonic seizure and complex partial seizure or the aura before primary generalised tonic – clonic seizure?
2. What is the type of seizure e.g. partial (focal) or generalized fit?
3. What is the behavior of the patient during the seizure e.g. motor (tonic – clonic, clonic, tonic, atonic, myoclonic, or absence) or sensory?
4. What is the duration of the seizure?
5. What are the provoking factors? (BOX 1 – 10)

BOX 1 – 10. Provocative factors for seizure

SEAL IN

Sleep deprivation	Flickering Lights, including TV and computer screens (primary generalised epilepsies only)
Physical and mental **E**xhaustion	
Alcohol (particularly withdrawal)	Intercurrent **IN**fections and metabolic disturbances

6. What are the associated symptoms during the convulsion e.g. cyanosis, urination, defecation, tongue biting?
7. What are the events that follow the convulsion e.g. headache, confusion, myalgia, or amnesia?
8. Are there features of pseudo-seizures?

CLINICAL CLUE

Status epilepticus is defined arbitrarily as seizures that continue for more than 30 minutes without ceasing spontaneously, or which recur so frequently that full consciousness is not restored between successive episodes.

EXAMPLES

The condition started suddenly as jerking movement involving the right side of the body whilst the patient was standing washing his face made him fallen to the ground. The patient developed jerking movement in the form of periodic relaxation and contraction of that part of the body lasting five minutes before the attacks is abolished spontaneously. During this attack the patient had not lost his consciousness, not urinate upon himself, and there was no tongue biting. After the attack the patient was just terrible from these events but fully conscious, able to respond to environment and asked for

help as he had slight weakness involving the right upper and lower limbs. No drugs or alcohol had been taken prior to this attack.

What is the diagnosis?
The diagnosis is simple partial motor seizure of clonic type.

The condition started whilst the patient was standing washing his face as stiffening and rigidity involving the right arm and the right leg made the patient fallen to the ground. The attack lasted five minutes and then abolished spontaneously. During this attack the patient had not lost his consciousness, not urinate upon himself, and there was no tongue biting. After the attack the patient was just terrible from these events but fully conscious, able to respond to environment and asked for help as he had slight weakness involving the right upper and lower limbs. No drugs or alcohol had been taken prior to this attack.

What is the diagnosis?
The diagnosis is simple partial motor seizure of tonic type.

The condition started suddenly whilst the patient was standing washing his face as instantaneous electric sensation involving the right arm and the right leg that was so unpleasant and lasting two minutes before the attacks is abolished spontaneously. During this attack the patient had not lost his consciousness, not urinate upon himself, and there was no tongue biting. After the attack the patient was just terrible from these events but fully conscious, able to respond to environment and asked for help. No drugs or alcohol had been taken prior to this attack.

What is the diagnosis?
The diagnosis is simple partial sensory seizure.

The condition started suddenly whilst the patient was sitting as abnormal sensation though to arise from the patient's abdomen that lasted for about 20 seconds before the patient lost his consciousness as the witness repeatedly called him with no response. The witness then had stated that the patient become motionless stare and stopped speech and movement for a while before he started to have repeated picking movements of his hands lasted for two minutes. During this attack the patient had not urinated upon himself and there was no tongue biting. After the attack the patient regained his consciousness gradually, feeling so terrible from these events and mild weakness involving the whole body. No drugs or alcohol had been taken prior to this attack.

What is the diagnosis?
The diagnosis is complex partial seizure.

The condition started suddenly whilst the patient was sitting as abnormal sensation though to arise from the patient's abdomen that lasted for about 20 seconds before the patient fallen to the ground lost his consciousness as the witness repeatedly called him with no response. The witness then had stated that the patient whole body became very stiff and rigid for 30 seconds. During this part, the patient lost his consciousness, his respiration is arrested, his tongue was severely injured, and he became markedly blue. Then he developed jerking movement with periodic relaxation and contraction involving the whole body parts for further 30 seconds; the jerking movement became less noticeable with the time until he became flaccid. After the attack the patient was urinated upon himself and his mouth was full with secretion. The patient stayed with disturbed consciousness for 30 minutes before he regained his consciousness so slowly, feeling so terrible from these events and weakness involving the whole body. No drugs or alcohol had been taken prior to this attack.

What is the diagnosis?
The diagnosis is secondary generalised tonic – clonic seizure.

18 RAJOOJ'S CLINICAL SKILLS

The condition started suddenly whilst the patient was sitting as sudden fall onto the ground with loss of consciousness as the witness repeatedly called him with no response. The witness then had stated that the patient whole body became very stiff and rigid for 30 seconds. During this part, the patient lost his consciousness, his respiration is arrested, his tongue was severely injured, and he became markedly blue. Then he developed jerking movement with periodic relaxation and contraction involving the whole body parts for further 30 seconds; the jerking movement became less noticeable with the time until he became flaccid. After the attack the patient was urinated upon himself and his mouth was full with secretion. The patient stayed with disturbed consciousness for 30 minutes before he regained his consciousness so slowly, feeling so terrible from these events and weakness involving the whole body. No drugs or alcohol had been taken prior to this attack.

What is the diagnosis?
The diagnosis is primary generalised tonic – clonic seizure.

The condition started suddenly whilst the patient was standing washing his face as stiffening and rigidity involving the whole body parts made the patient collapsed and fallen to the ground. During the attack which last one minute, the patient lost his consciousness, his respiration is arrested, he became markedly blue, and his tongue is severely bitten. He did not develop jerking movement with periodic relaxation and contraction after that. The patient after that had urinated upon himself, was so terrible from these events, unable to respond to environment, and felt headache and would like to sleep. No drugs or alcohol had been taken prior to this attack.

What is the diagnosis?
The diagnosis is primary generalised tonic seizure.

The condition started suddenly whilst the patient was standing washing his face as jerking movement with periodic relaxation and contraction involving the whole body parts made the patient collapsed and fallen to the ground. During the attack which last one minute, the patient lost his consciousness; the jerking became less noticeable with the time till the patient became flaccid. The patient after that had urinated upon himself, was so terrible from these events, unable to respond to environment, and felt headache and would like to sleep. No drugs or alcohol had been taken prior to this attack.

What is the diagnosis?
The diagnosis is primary generalised clonic seizure.

The condition started suddenly whilst the patient was sitting as sudden fall onto the ground with loss of consciousness as the witness repeatedly called him with no response. The witness then had stated that the patient whole body became very stiff and rigid for 30 seconds. During this part, the patient lost his consciousness, his respiration is arrested, his tongue was severely injured, and he became markedly blue. Then he developed jerking movement with periodic relaxation and contraction involving the whole body parts for further 30 seconds; the jerking movement became less noticeable with the time until he became flaccid. After the attack the patient was urinated upon himself, his mouth was full secretion, and had severe difficulty in spontaneous breathing. The patient had not regain consciousness for further 3 minutes and again developed stiffening and shaking period whilst rushed by his brother, the witness, to the hospital. The patient in his way to the hospital did not regain consciousness and developed further two attacks; each lasted 5 minutes. In the hospital the medical staff.....

What is the diagnosis?
The diagnosis is status epilepticus of tonic clonic type.

FEVER

Heat and cold sensitive neurons are located partly in the anterior hypothalamus. The posterior hypothalamus integrates signals from the anterior hypothalamus with temperature information from peripheral receptors and then modulates the body's heat production, conservation and loss. According to the studies of healthy individuals 18-40 years of age, the mean oral temperature is 36.8 ± 0.4°C, with low levels at 6 a.m. and higher level at 4-6 p.m. A regulated elevation in the body temperature above the customary set point of the hypothalamic thermostat is called fever. Pyrexia (fever) of unknown origin (PUO or FUO) was defined by Petersdorf and Beeson in 1961 as:

(1) Temperature of >38.3 °C on several occasions.
(2) Duration of fever of >3 weeks.
(3) Failure to reach a diagnosis despite one week of inpatient investigation.

Causes of PUO are outlined in BOX 1 – 11.

BOX 1 – 11. Causes of pyrexia of unknown origin

Infections	Autoimmune hypersensitivity diseases	Familial – hereditary diseases
(1) Bacterial diseases Systemic infection:- • Tuberculosis • Brucellosis • Salmonellosis Localized infections:- • Pneumonia • Pyelonephritis (2) Viral diseases like cytomegalovirus, infectious mononucleosis and hepatitis (3) Fungal diseases like histoplasmosis and coccidioidomycosis (4) Parasitic diseases like giardiasis, toxoplasmosis, and malaria	Rheumatoid arthritis Rheumatic fever Systemic lupus erythematosus	Familial Mediterranean fever Hypertriglyceridemia
	Neoplasms	Thermoregulatory disorders
	Leukemia Lymphoma	Brain tumor Cerebrovascular accident Hyperthyroidism Pheochromocytoma
	Granulomatous disease	Miscellaneous
	Sarcoidosis Crohn's disease	Diabetes insipidus Factitious fever Pulmonary embolism

In febrile disorders, there is a disturbance of heat regulation, so that thermostatic mechanism controlling heat gain and loss is set at a level higher than normal. While temperature is rising to this new level, the skin vessels are constricted so that the body feels cold, and the patient may even shiver violently, this shivering is called a rigor. When the higher temperature is reached, heat loss again becomes apparent; the skin vessels dilate and the body feels warm and sweaty. Certain patterns of fever are recognized as shown in BOX 1 – 12.

Feeling cold and shivering both accompanies a rising temperature, while feeling hot and sweating accompany a falling temperature. Feelings of heat and sweating also accompany menopause. Normally the body temperature rises during the day and falls during the night. When fever exaggerates this swing, night sweats occur. Night sweats occur in tuberculosis and malignancy.

BOX 1 – 12. Patterns of fever

Continued fever is a type of fever that is fluctuating but not more than 1°C during 24 hours and never reaching the normal base – line. It occurs in typhoid fever, drug fever, malignant hyperthermia		
Remittent fever is similar to continued fever but with a daily fluctuation of more than 2 °C and temperature does not return to normal. This type of fever is not characteristic of any particular disease		
Intermittent fever is a type of fever that is present only during few hours in 24 hours i.e. temperature falls to normal each day. It occurs in pyogenic infections, lymphomas, miliary tuberculosis		
Relapsing fever is characterized by returning of temperature to normal for days before rising again. It occurs in:		
1	Malaria	Tertian-3-day pattern, fever peaks every other day (Plasmodium vivax, P. ovale) Quartan-4-day pattern, fever peaks every 3rd day (P. malariae)
2	Lymphoma	Pel-Ebstein fever (1 week with fever and 1 week without fever) of Hodgkin's disease (rare)
3	Pyogenic infection	Similar to intermittent fever but had wider fluctuation. It is called Hectic fever

HOW TO APPROACH TO FEVER?

SOCRATES

S: Subjective or objective O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Duration and Course) E: Exacerbating and precipitating factors S: Severity

PRESENTATION: FEVER

1. Is the fever subjectively felt by the patient or it is genuine measured by a thermometer?
2. What is the mode of onset e.g. sudden or gradual?
3. What is the character of the fever e.g. continuous, intermittent, or remittent?
4. What are the relieving factors e.g. cool sponging or medications?
5. What are the associated symptoms e.g. sweating, chills, or rigor? If there is sweating determine the time of occurrence (day or night time) and its nature whether profuse (drenching) or slight. Chills mean just bed shaking and are encountered in most febrile illnesses. However, a teeth-chattering, bed-shaking chill indicative of a true rigor is usually associated with bacteremia, pyelonephritis, abscess and biliary tract infection. Malaise, headache, and pain in the muscles and joints often accompany fever.
6. What is the timing of fever e.g. duration and course? Duration means the time since fever first noticed and the course means the progression of fever whether it started low grade then built up to be high grade or the fever was of low grade from the start.
7. What are the exacerbating and precipitating factors e.g. drugs or heat whether?
8. What is the severity of fever e.g. high, moderate or low grade? High grade fever made the patient unable to perform any activity, interfered with sleeping and eating and the patients are usually kept in bed.

CLINICAL CLUE

DEFINITIONS USED TO DESCRIBE THE CONDITION OF SEPTIC PATIENTS

Bacteremia means the presence of bacteria in blood, as evidenced by positive blood cultures. Septicemia means the presence of microbes or their toxins in blood. Systemic inflammatory response syndrome (SIRS) means the presence of two or more of the following conditions:

1. Fever (oral temperature >38°C) or hypothermia (<35°C).
2. Tachypnea (>24 breaths/min).
3. Tachycardia (heart rate >90 beats/min).
4. Leukocytosis (>12,000/μL), leukopenia (<4,000/μL), or >10% bands.

Sepsis means SIRS that has a proven or suspected microbial etiology. Septic shock means sepsis with hypotension (systolic blood pressure <90 mmHg, or 40 mmHg less than patient's normal blood pressure) for at least 1 h of adequate fluid resuscitation or need for vasopressors to maintain systolic blood pressure ≥90 mmHg. Refractory septic shock means septic shock that lasts for >1 h and does not respond to fluid or pressor administration. Multiple-organ dysfunction syndrome means dysfunction of more than one organ, requiring intervention to maintain homeostasis.

EXAMPLE

The condition started as a gradual development of low grade fever for three days that sooner built up to be high grade made the patient unable to perform daily activities and kept him in bed, experienced mainly at night with periods of shivering. At the early hours of morning time, the patient experienced profuse sweating. No medications had been used by the patient as she felt better at day time, but at night time cool sponging had been applied to the face and the limbs which some what helped to improve the condition for certain time. This condition was associated with back pain that...

WHAT ARE THE ENDOCRINE CAUSES OF SWEATING?
Endocrine causes of sweating include thyrotoxicosis, pheochromocytoma, diabetes mellitus, acromegaly, and menopause.

WHAT ARE THE NON – INFECTIUOS CAUSES OF SWEATING?
Non-infective causes of sweating include alcohol misuse, anxiety, lymphoma, excessive heating of the environment, and endocrine causes.

GIVE US EXAMPLES OF DRUGS THAT CAN CAUSE FEVER
Drugs that cause fever include penicillins, cephalosporins, antituberculous, anticonvulsants (particularly phenytoin), and methyldopa.

HYPERTHERMIA

Although most patients with elevated body temperature have fever, there are circumstances in which elevated temperature represents not fever but hyperthermia (BOX 1 – 13).

BOX 1 – 13. Distinctions between fever and hyperthermia

FEVER	HYPERTHERMIA
Associated with raised thermostatic set point	Not associated with raised thermostatic set point
Responds to antipyretics	Not responds to antipyretics
Associated with sweating and the skin can be cold	Not associated with sweating and the skin is dry
Dantrolene is not indicated	Respond to dantrolene (according to the cause)

Hyperthermia is characterized by an uncontrolled increase in body temperature that exceeds the body's ability to lose heat and the setting of the hypothalamic thermoregulatory center is unchanged. Some causes of hyperthermia are shown in BOX 1 – 14.

BOX 1 – 14. Some causes of hyperthermia

MECHANISM	EXAMPLES
Heat stroke	Exercise in higher-than-normal heat and/or humidity
Drug	Salicylates, lithium
Neuroleptic malignant syndrome	Phenothiazines, metoclopramide, domperidone, and dopaminergic agents
Malignant hyperthermia	Inhalational anesthetics, succinylcholine
Endocrinopathy	Thyrotoxicosis, pheochromocytoma
CNS damage	Cerebral hemorrhage, status epilepticus, hypothalamic injury

CHEST PAIN

Chest pain is a common presentation of cardiac disease, but can also be a manifestation of anxiety or other conditions (BOX 1 – 15). A number of key characteristics help to distinguish ischemic cardiac pain from that of other causes. Typical chest pain has three components: (1) substernal chest pain or discomfort; (2) provoked by exertion or emotional stress; (3) relieved by rest and/or nitroglycerin. Atypical chest pain has two of the above three components for typical chest pain and non-anginal chest pain has none of the above components for typical chest pain.

BOX 1 – 15. Differential diagnosis of chest pain

Central		Peripheral	
Cardiac		Lungs/pleura	
Myocardial ischemia (angina)	Myocarditis	Pulmonary infarct	Malignancy
Myocardial infarction	Pericarditis	Pneumonia	Tuberculosis
	Mitral valve prolapse	Pneumothorax	Connective tissue disorders
Esophageal		Neurological	
Esophagitis	Mallory-Weiss syndrome	Prolapsed intervertebral disc	Thoracic outlet syndrome
Esophageal spasm		Herpes zoster	
Massive pulmonary embolus		Musculoskeletal	
		Osteoarthritis	
Mediastinal		Costochondritis (Tietze's syndrome)	
Tracheitis	Malignancy	Rib fracture/injury	
		Intercostal muscle injury	
Aortic		Epidemic myalgia (Bornholm disease)	
Aortic dissection	Aortic aneurysm		
Anxiety/emotion			

Patients with ischemic heart disease fall into two large groups: patients with chronic coronary artery disease (CAD) who most commonly present with stable angina and patients with acute coronary syndromes (ACSs). The latter group, in turn, is composed of patients with acute myocardial infarction (MI) with ST-segment elevation on their presenting electrocardiogram (STEMI) and those with unstable angina and non-ST-segment elevation myocardial infarction (UA/NSTEMI). Stable angina is an effort (exertion) related chest pain due to fixed atheromatous stenosis of one or more coronary arteries. Unstable angina is a clinical syndrome that is characterized by a new onset angina, rapidly worsening angina (crescendo angina), angina on minimal exertion, angina on rest and post-infarct angina. Variant angina (prinzmetal's angina) is a spontaneous transient ST segment elevation on the ECG with the onset of anginal pain. Microvascular angina means microvascular dysfunction without detectable lesions or spasm in the large

coronary artery. Pseudonormalized angina means patient who's resting ECG shows T waves inversion that normalized during angina. Postprandial angina means development of angina after heavy meals. Warm up angina (walk through angina) is the term applied for some patients who find that anginal discomfort comes when they start walking and that later it does not recur despite regular effort. Decubitus angina means angina on lying flat. Nocturnal angina means development of angina after vivid dreams. Angina equivalent refers to symptoms of myocardial ischemia other than anginal pain. These include dyspnea, nausea, fatigue, and faintness, and are common in women, in elderly and in diabetics. Cardiac syndrome X refers to those patients with a good history of angina, a positive exercise test and angiographically normal coronary arteries. The syndrome is more common in women than in men.

IN THE ABSENCE OF CORONARY ATHEROSCLEROSIS WHAT ARE THE DISORDERS CAUSING ANGINA?

Aortic stenosis, aortic regurgitation, pulmonary hypertension, and hypertrophic cardiomyopathy may cause angina in the absence of coronary atherosclerosis.

WHAT IS MEANT BY PRIMARY AND SECONDARY UNSTABLE ANGINA?

Distinguishing primary from secondary unstable angina is of clinical value. Acute worsening of a coronary stenosis causes **primary unstable angina** by limiting coronary blood flow. **Secondary unstable angina** arises as a consequence of increased myocardial oxygen demand superimposed on severe underlying coronary disease. The conditions with the potential to provoke secondary unstable angina include tachyarrhythmia, fever, hypoxia, anemia, hypertensive crisis, and thyrotoxicosis. Secondary unstable angina should resolve with successful treatment of the precipitating condition.

WHAT ARE THE ASSOCIATIONS OF PRINZMETAL'S ANGINA

1. Migraine.
2. Raynaud's phenomenon.
3. Aspirin-induced asthma.

PRESENTATION: CHEST PAIN

1. Where is the site of chest pain e.g. central or peripheral? Cardiac pain is typically located in the center of the chest because of the derivation of the nerve supply to the heart and mediastinum.

CLINICAL CLUE

When a patient with an anginal pain is asked to localize the sensation, he or she will typically place their hands over sternum, sometimes with a clenched fist (Levine's sign).

2. What is the onset of the chest pain? The pain of myocardial infarction typically takes several minutes or even longer to develop; similarly, angina builds up gradually in proportion to the intensity of exertion. Pain that occurs after rather than during exertion is usually musculoskeletal or psychological in origin. The pain of aortic dissection, massive pulmonary embolism or pneumothorax is usually very sudden or instantaneous in onset.
3. What is the character of the chest pain? Cardiac pain is typically dull, constricting, choking or 'heavy', and is usually described as squeezing, crushing, burning or aching but not sharp, stabbing, pricking or knife-like. The sensation can be described as breathlessness. Patients often emphasize that it is a discomfort rather than a pain.
4. What is the radiation of the chest pain? Ischemic cardiac pain, especially when severe, may radiate to the neck, jaw, and upper or even lower arms. It is rarely localized below the umbilicus or above the mandible. Chest discomfort due to myocardial ischemia does not radiate to the trapezius muscles; such a radiation pattern is more typical of pericarditis. Occasionally, cardiac pain may be experienced only at the sites of radiation or in the back. Pain situated over the left anterior chest and radiating laterally may have many causes, including pleural or lung disorders, musculoskeletal problems and anxiety.
5. What are the associated symptoms? The pain of myocardial infarction, massive pulmonary embolism or aortic dissection is often accompanied by autonomic disturbance including sweating, nausea and vomiting. Breathlessness, due to pulmonary congestion arising from transient ischemic left ventricular dysfunction, is often a prominent and occasionally the dominant feature of myocardial infarction or angina (angina equivalent). Breathlessness may also accompany any of the respiratory causes of chest pain and may be associated with cough, wheeze or other respiratory symptoms. Classical gastrointestinal symptoms (esophageal reflux, esophagitis, peptic ulceration or biliary disease) may provide the clue to the source of non-cardiac chest pain but effort-related 'indigestion' is usually due to heart disease.

6. What is the timing of chest pain? Although ischemia may commence at any time of the day or night, circadian variations have been reported such that clusters are seen in the morning within a few hours of awakening. Stable anginal pain usually lasts <2–10 min, unstable angina pain usually lasts <20 min, and myocardial infarction pain usually lasts ≥30 min but it is variable. The combination of substernal chest pain persisting for >30 min and diaphoresis (profuse sweating) strongly suggests STEMI.
7. What are the exacerbating and the precipitating factors of chest pain? Anginal pain occurs during (not after) exertion and is promptly relieved (in less than 5 minutes) by resting. The pain may also be brought on or exacerbated by emotion and tends to occur more readily during exertion, after a large meal or in a cold wind. In crescendo or unstable angina, similar pain may be precipitated by minimal exertion and may occur at rest. The increase in venous return or preload induced by lying down may also be sufficient to provoke pain in vulnerable patients (decubitus angina). In contrast, pleural or pericardial pain is usually described as a 'sharp' or 'catching' sensation that is exacerbated by breathing, coughing or movement. Pain associated with a specific movement (bending, stretching, turning) is likely to be musculoskeletal in origin.
8. What are the factors relieving chest pain? Anginal pain is relieved by rest and sublingual glyceryl trinitrate (GTN).
9. What is the severity of chest pain? The severity is assessed as to whether the pain interferes with walking, talking, sleeping, and eating.

EXAMPLES

The condition repeatedly started gradually over few minutes as a moderately severe chest pain that was located in the center of the chest when the patient walked more than 300 meters. Each time the patient felt it as burning pain, lasted 5 minutes, radiated to the jaw and left shoulder, and associated with mild sweating and nausea. The pain however soon relieved with rest and sublingual glyceryl trinitrate (GTN).

What is the diagnosis?
The most likely diagnosis is stable angina.

What is the diagnosis if such a pain presents at rest or more severe than usual pain or occurs at lesser degree of exertion or at rest?
The diagnosis is unstable angina if such a pain presents at rest or is more severe than usual pain or occurs at lesser degree of exertion or at rest.

The condition started gradually as a severe chest pain that occurred after heavy meal intake last night. The pain was located in the center of the chest, squeezing in nature, radiated to the neck and inner border of the left arm, and was accompanied by nausea, two attacks of vomiting, and transient breathlessness. The pain experienced for the first time, persisted for 30 minutes and relieved slightly by resting.

What is the diagnosis?
The most likely diagnosis is myocardial infarction.

The condition started suddenly as chest pain that was located in the center of the chest slightly to the left of the sternum, sharp in nature and catch the patient during coughing and inspiration, radiated into the neck, arms, and left shoulder, severe in intensity but varies with movement and respiration, relieved by sitting up, and intensified by lying supine. The chest pain was preceded by low grade fever for the previous one week and was accompanied by nausea and awareness of heart beats.

What is the diagnosis?
The most likely diagnosis is acute pericarditis.

The condition started very suddenly as severe chest pain that was located in the upper part of the chest in the center, sharp and tearing in nature, felt penetrating through to the back in the areas between the scapulae, very severe in intensity especially at the start, and relieved only when seek medical advice and given injectable form of medication. The chest pain was associated with

profuse sweating and breathlessness. The pain was later felt in the back and migrating down with the time. This is the first time the patient felt such a pain.

What is the diagnosis?
The most likely diagnosis is aortic dissection.

The condition started two days ago as low grade fever, sour throat, and dry cough. The condition after that developed gradually as chest pain that was located in the left of the chest anteriorly, persistent, sharp and knifelike in nature, sometimes radiated to the left side of the abdomen and back, severe in intensity, precipitated by breathing, coughing, and movements of the trunk, and relieved by lying on the involved left side. The chest pain was associated with sense of breathlessness, and productive cough.

What is the diagnosis?
The most likely diagnosis is pleural pain from inflammation of the parietal pleura, as from pleurisy, pneumonia, pulmonary infarction, or neoplasm.

The condition started gradually over the preceding one month as occasional attacks of chest pain that was located in the center of the chest, variable in time, burning in nature, sometimes radiated to the back, variable from mild to severe in intensity, precipitated by large meals and bending or lying down, and relieved by antacids medication and belching. The chest pain was associated with regurgitation of sour material, no sweating or breathlessness. This is the first time the patient felt such a pain.

What is the diagnosis?
The most likely diagnosis is esophageal pain e.g. gastro-esophageal reflux disease (GERD).

The condition started gradually over the preceding two days as chest pain that was variable in location but often below the left breast and along the costal cartilages, variable in time from few minutes to one hours, stabbing in nature, not radiated, mild to moderate in intensity, aggravated by movement of the chest, trunk, and arms, and relieved by pain medications.

What is the diagnosis?
The most likely diagnosis is musculoskeletal chest pain.

Esophageal pain can mimic that of angina in that it is precipitated by exercise and may be relieved by nitrates. Musculoskeletal chest pain is sometimes accompanied by local tenderness over ribs or costal cartilage. However ischemic chest pain can also accompanied by local tenderness over a rib or costal cartilage. Pericardial chest pain may be preceded by low-grade fever in contrast to myocardial infarction, in which the pain precedes the fever. Status anginosus refers to severe angina that has reached a chronic phase and that is refractory to treatment; it is usually associated with extensive atherosclerosis not amenable to reperfusion.

WHAT ARE THE CAUSES OF MYOCARDIAL INFARCTION?
Myocardial infarction may be caused by coronary atherosclerosis (most commonly), coronary emboli, coronary vasospasm, congenital coronary anomalies, coronary vasculitis, infiltrative and degenerative vascular disease, and trauma.

PALPITATION

Palpitation means an unexpected awareness of the heart beating in the chest. Patients experience a rapid, forceful or irregular cardiac impulse and describe this as thumping, pounding, fluttering, jumping, racing or skipping. Palpitation may occur in the following clinical settings:

- Sinus rhythm with anxiety (sinus tachycardia). Healthy people are occasionally aware of their heart beating when they have normal (sinus) rhythm. This is often related to physical or psychological stress: for example, after exercise, or when waiting for an examination! It is more common in bed at night when external visual and auditory inputs are minimal and visceral sensations more prominent. Slim people may notice it when lying on their left side. Palpitation can also be brought on by excessive caffeine intake (tea, coffee, cola) and by nicotine from smoking.
- Intermittent irregularities of the heartbeat (atrial and ventricular ectopic beats). Ectopic beats (extrasystoles) are a benign cause of palpitation at rest and are abolished by exercise. Patients may describe 'missed beats', sometimes followed by a particularly strong heartbeat. This occurs because the ectopic beat produces a small stroke volume due to incomplete left ventricular filling. The subsequent compensatory pause leads to ventricular overfilling and a forceful contraction with the next normal beat.
- Abnormal rhythm i.e. arrhythmia (atrial fibrillation, supraventricular tachycardia, and ventricular tachycardia). Pathological rapid heartbeat (tachyarrhythmia) starts suddenly, lasts for minutes or hours, and is unrelated to anxiety or stress. The history helps distinguish different types of palpitation (BOX 1 – 16). In such a condition it is necessary to identify any underlying pathological process e.g. structural heart disease.

BOX 1 – 16. Descriptions of arrhythmias

Arrhythmia	Patient's description
Atrial fibrillation (AF)	Heart 'jumping about' or 'racing' Associated breathlessness May be asymptomatic
Supraventricular tachycardia (SVT)	Heart racing or fluttering Associated polyuria
Ventricular tachycardia (VT)	Heart racing or fluttering Associated breathlessness May present as syncope rather than as palpitation

HOW TO APPROACH TO LIMB ISCHEMIA?

S: Site O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Duration and Course) E: Exacerbating and precipitating factors S: Severity

SOCRATES

PRESENTATION: UNEXPECTED AWARENESS OF THE HEART BEATING

1. Is it positional? Palpitations that are positional may reflect a structural process within (e.g., atrial myxoma) or adjacent to (e.g., mediastinal mass) the heart.
2. What is the mode of onset and termination of palpitation? If the patient complains of a rapid heartbeat, it is important to find out whether the palpitations are of sudden or gradual onset and offset. Cardiac arrhythmias are usually instantaneous in onset and offset, whereas the onset and offset of sinus tachycardia is more gradual.
3. What is the character e.g. regular or irregular, fast or slow (ask the patient to tap it out)? A completely irregular rhythm is suggestive of atrial fibrillation, particularly if it is rapid. It may be helpful to ask the patient to tap the rate and rhythm of the palpitations with his or her finger.
4. What are the relieving factors e.g. breathe holding, exercise, spontaneous? Patients may have learned maneuvers that will return the rhythm to normal. Attacks of supraventricular tachycardia may be suddenly terminated by increasing vagal tone with the Valsalva maneuver, carotid massage, by coughing, or by swallowing cold water or ice cubes.
5. What are the associated symptoms e.g. chest pain, dyspnea, polyuria, syncope? The awareness of rapid palpitations followed by syncope suggests ventricular tachycardia.
6. What is the timing of the attacks e.g. duration of each attack and the course of the attacks?
7. What are the precipitating and exacerbating factors e.g. exercise, alcohol, coffee, tea, or cola, drugs, anxiety, or stress?
8. What is the severity of the attack?

EXAMPLES

The condition repeatedly started gradually over few minutes as unexpected awareness of heart beats that were regular, so fast, and unrelated to specific positions. Each time the attack lasted 20 minutes and gradually disappeared in a spontaneous manner. The attack was repeatedly recurred once the student awaiting her exam and disappeared after around 10 minutes once she engaged into the exam. The student had noticed no increase in urine volume during the attack and each attack was not followed by faintness or lightheadedness.

What is the diagnosis?
The most likely diagnosis is sinus tachycardia

The condition repeatedly occurred whilst the patient at rest or went to bed as unexpected awareness of heart beats that were irregular, infrequent, and unrelated to specific positions. The Patient initially felt missed beat as if his heart stopped what it was doing but was followed by a particularly strong heart beat. The attack occurred repeatedly but infrequently and disappeared when the patient started to walk or to exercise. The patient had noticed no increase in urine volume during these events and was not followed by faintness or lightheadedness. The patient lastly was heavy cola, tea, and coffee consumer.

What is the diagnosis?
The most likely diagnosis is ectopic beats

The condition repeatedly started suddenly as unexpected awareness of heart beats that were regular, so fast described by the patient as that my heart is fluttering, and unrelated to specific positions. The attack lasted 20 minutes and disappeared suddenly when the patient used to introduce his right index finger into his mouth in a way of induction of vomiting. The patient had noticed an increase in urine volume during the attack and each attack was accompanied by a sense of breathlessness but no loss of consciousness. The attack happened twice monthly despite regular medicine intake described by the patient's physician.

What is the diagnosis?
The most likely diagnosis is supraventricular tachycardia (SVT)

The condition started suddenly as unexpected awareness of heart beats that were completely irregular, so fast, and unrelated to specific positions. The attack was described by the patient as that the heart is jumping and persisting up to the time of history taking. The attack was accompanied by a sense of breathlessness and faintness that gradually faded with the time. The patient lastly was heavy cola, tea, and coffee consumer.

What is the diagnosis?
The most likely diagnosis is atrial fibrillation (AF)

The condition started suddenly as unexpected awareness of heart beats that were regular and unrelated to specific positions. The attack was described the patient as that the heart is fluttering too fast, persisting for around 5 minutes and was accompanied by a sense breathlessness till he lost his consciousness.

What is the diagnosis?
The most likely diagnosis is ventricular tachycardia (VT)

HOW TO PERFORM CAROTID SINUS MASSAGE (FOR RESIDENT DOCTORS)?

1. Ensure there is no significant carotid artery disease (carotid bruits) via carotid auscultation.
2. Provide continuous electrocardiographic monitoring.
3. Patient is in supine position with the head slightly extended.
4. Start with right carotid sinus massage.
5. Apply firm rotary pressure to the carotid artery at the level of the third cervical vertebra for 5 seconds. Alternatively, steady pressure can be applied.
6. If no response, massage left carotid sinus.
7. Generally, right carotid sinus massage decreases the sinus node discharge, and left carotid sinus massage slows atrioventricular conduction.
8. Do not massage both carotid sinuses at the same time.
9. Single application of carotid sinus pressure may be effective in about 20–30% of patients with paroxysmal supraventricular tachycardias; multiple applications can terminate tachycardia in about 50% of patients.
10. Asystole is a potential but rare complication.

LIMB ISCHEMIA

The legs are eight times more commonly affected than the arms because arterial supply to the legs is less well developed in relation to the muscle mass and because the lower limb is more frequently affected by atherosclerosis. There are four stages of lower limb ischemia. These are:

1. Asymptomatic stage. Most patients in this stage are asymptomatic because they choose not to walk very far or because their exercise tolerance is limited by other pathology.
2. Intermittent claudication (discussed below).
3. Night/rest pain. The patient goes to bed and falls asleep but is woken 1-2 hours later by severe pain in the foot, usually in the instep. This occurs because the beneficial effects of gravity on lower limb perfusion are lost on lying down. Sleep is also associated with a reduction in heart rate, blood pressure and cardiac output. Patients often obtain relief by hanging the leg out of bed or by getting up and walking around but, when they return to bed, symptoms recur. The pain and sleep disturbance may be so debilitating that they sleep in a chair. This leads to dependent edema, the increased interstitial tissue pressure causing further reduction in tissue perfusion and more pain. Rest pain usually indicates severe, multi-level arterial disease.

CLINICAL CLUE

In diabetic patients with rest pain it may be difficult to differentiate between an arterial cause and diabetic neuropathy since both may be worse at night. Neuropathic pain is not, however, usually confined to the foot, is associated with burning and tingling, is not relieved by dependency.

4. Tissue loss (ulceration/gangrene). In patients with critical limb ischemia, trivial injuries do not heal, allowing bacteria to enter, leading to gangrene and/or ulceration. Without revascularization, the ischemia rapidly progresses, leading to amputation and/or death.

CLINICAL CLUE

Critical limb ischemia is defined as rest (night) pain, requiring opiate analgesia, and/or tissue loss (ulceration or gangrene), present for more than 2 weeks, in the presence of an ankle blood pressure of less than 50 mmHg. Rest pain and no tissue loss with ankle pressures above 50 mmHg is sometimes known as subcritical limb ischemia. The term severe limb ischemia (SLI) is often used to describe both entities. Whereas intermittent claudication is usually due to single-segment plaque, critical limb ischemia is always due to multi-level disease.

INTERMITTENT CLAUDICATION

Intermittent claudication describes pain felt in the legs on walking. It is divided into three types; arterial, venous, and neurogenic (BOX 1 – 17).

BOX 1 - 17. The clinical features of arterial, neurogenic and venous claudication

	Arterial	Neurogenic	Venous
Pathology	Stenosis or occlusion of major lower limb arteries	Lumbar nerve root or cauda equina compression (spinal stenosis)	Obstruction to the venous outflow of the leg due to iliofemoral venous occlusion
Site of pain	Muscles, usually the calf but may involve thigh and buttocks	Ill-defined. Whole leg. May be associated with numbness and tingling	Whole leg. 'Bursting' in nature
Laterality	Unilateral if femoropopliteal, and bilateral if aorto-iliac disease	Often bilateral	Nearly always unilateral
Onset	Gradual after walking the 'claudication distance'	Often immediate on walking or standing up	Gradual, from the moment walking starts
Relieving features	On stopping walking, the pain disappears completely in 1-2 minutes	Bending forwards and stopping walking. May sit down for full relief	Leg elevation
Color	Normal or pale	Normal	Cyanosed. Often visible varicose veins
Temperature	Normal or cool	Normal	Normal or increased
Edema	Absent	Absent	Always present
Pulses	Reduced or absent	Normal	Present but may be difficult to feel owing to edema
Straight leg raising	Normal	May be limited	Normal

Arterial intermittent claudication is pain felt in the legs on walking due to arterial insufficiency. It is the most common symptom of peripheral arterial diseases. The pain typically occurs in the calf secondary to femoropopliteal disease but may be felt in the thigh and/or buttock if proximal (aorto-iliac) obstruction to blood flow is present. Patients describe a tightness or 'cramp-like' pain which develops after a relatively constant distance, which is shorter if walking uphill. The pain disappears completely within a few minutes of rest but recurs on walking. The claudication distance is how far patients say they can walk before pain starts. Patients often underestimate this. In neurogenic claudication, there is leg pain on walking due to neurological and musculoskeletal disorders of the lumbar spine. Venous claudication describes pain due to venous outflow obstruction from the leg following extensive deep vein thrombosis. Neurogenic and venous claudication is much less common than arterial claudication, and can be distinguished on history and examination.

HOW TO APPROACH TO LIMB ISCHEMIA?

S: Site O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Duration and Course) E: Exacerbating and precipitating factors S: Severity

SOCRATES

PRESENTATION: LIMB PAIN ON WALKING

1. Where is the site e.g. leg (one or both) or arm?
2. What is the onset of the attack?
3. What is the character e.g. cramping, ill-defined, or bursting?
4. What are the relieving factors e.g. bed hanging, walking, leg elevation, stooping forward, stop walking?
5. What are the associated symptoms e.g. feeling of coldness, numbness, or pallor?
6. What is the timing of the attack (duration and course)?
7. What are the exacerbating and precipitating factors e.g. walking (determine the distance) or standing?
8. What is the severity of the attack?

EXAMPLES

The condition started gradually as a severe cramping pain that involve the left calf muscles when the patient walked to more than about 500 meters. This pain rendered the patient unable to continue walking and so he stopped walking with the result of prompt relief in less than one minute. The condition was however soon recurred once walking for further 500 meters was resumed.

The patient had noticed no rest/night foot pain or swelling of the left leg but had noticed that ascent for only one flight stairs brought up the same pain.

What is the diagnosis?
The diagnosis is arterial intermittent claudication due to left femoropopliteal circulation ischemia.

The condition started gradually as a severe cramping pain that involve the buttock muscles when the patient walked to more than about 100 meters. This pain rendered the patient unable to continue walking and so he stopped walking with the result of prompt relief in less than two minutes. The condition was however soon recurred once walking for further 100 meters was resumed. The patient had noticed no rest/night foot pain or swelling of the legs but had experienced that lesser distance might produce the same pain when he ascent up.

What is the diagnosis?
The diagnosis is arterial intermittent claudication due to aorto-iliac circulation ischemia.

The condition started immediately as an ill-defined severe pain involving both legs when the patient stood up or started walking. This pain was accompanied by pins and needles sensation rendered him unable to continue walking and so he stopped walking and bended foreword with the result of pain relief although in the last days he had to stop walking and sit down to get relief. The condition was however soon recurred once standing was resumed or walking was started. The patient had noticed no rest/night foot pain or swelling of the legs but had experienced that descent from stairs produced the same pain.

What is the diagnosis?
The diagnosis is neurogenic intermittent claudication.

The condition started gradually as a mild bursting pain involving the whole left leg from the moment the patient started walking. The pain then built up to reach higher intensity with continuing walking so that she stopped walking and sat down to get relief although such an attempt was not always successful and the patient had to lie down and raise her left leg when isolated. The condition was however soon recurred once walking was resumed again. The patient had noticed that her left leg is too blue in color and even mildly swollen but there was no rest/night pain when she used to lie flat and elevate her left leg.

What is the diagnosis?
The diagnosis is venous intermittent claudication.

The condition started gradually as a severe foot pain 1 – 2 hours after sleeping that awoke the patient from sleep. In the early course of his illness he had to hang his leg out of his bed to get some sort of gradual relief but his condition had deteriorated to the degree that such maneuver had not resulted in pain relief. He tried then to walk for few minutes around his bed when the pain started; he got some pain relief with this maneuver. Unfortunately however he stayed to have pain even with such a trial that made him slept in a chair rather than in a bed to get some pain relief. Days after that he experienced foot pain not only at night but also with leg hanging, walking around for few minutes, with chair sleeping, and lastly even at rest.

What is the diagnosis?
The diagnosis is rest/night arterial ischemic pain.

FEET/ANKLE/LEGS SWEELING

Tissue spaces are divided into intracellular spaces and extracellular spaces. Extracellular space is composed of intravascular and interstitial compartments. Edema is defined as accumulation of fluid in the interstitial spaces.

CLINICAL CLUE

Anasarca refers to gross, generalized edema. Ascites and hydrothorax refer to accumulation of excess fluid in the peritoneal and pleural cavities, respectively, and are considered to be special forms of edema.

The distribution of water between the vascular and interstitial spaces is determined by the balance between hydrostatic pressure, which forces water out of the capillary, and colloid osmotic (oncotic) pressure, which sucks fluid into the vascular space (Starling's forces). Oncotic pressure is largely dependent on circulating protein concentration, particularly serum albumin. Edema can be generalized, localized, postural, or idiopathic. Two principal causes of generalized edema are fluid overload and hypoproteinemia. Distinguishing them is performed by assessing the jugular venous pulse (JVP). The JVP is usually elevated in fluid overload but not in hypoproteinemia. Fluid overload may be due to heart failure or renal disease, or from iatrogenic causes. Iatrogenic causes result from excessive fluid replacement, especially intravenously, which produces fluid overload. Hypoproteinemia, particularly hypoalbuminemia, reduces oncotic pressure and encourages fluid to move to the interstitial space, thus causing edema. Hypoalbuminemia results from nephrotic syndrome (increased loss), liver failure (decreased synthesis), malnutrition, and malabsorption. When edema is generalized, its distribution is determined by gravity. In ambulant patients, edema first appears at the ankles and over the dorsum of the foot. In bed-bound patients, edema often appears first over the sacrum. If the patient lies flat, it may involve the face and hands. Localized edema may be caused by venous, lymphatic (lymphedema), inflammatory or allergic disorders. Any increased venous pressure will increase hydrostatic pressure within capillaries, producing edema in the area drained by that vein. Venous causes include deep vein thrombosis, external pressure from a tumour or pregnancy, or valvular incompetence from previous thrombosis or surgery. Conditions which impair the normal pumping action of muscles e.g. hemiparesis and forced immobility (air travel) increase venous pressure by impairing venous return. Normally, interstitial fluid returns to the central circulation via the lymphatic system. Any cause of impaired lymphatic flow, e.g. intraluminal or extraluminal obstruction, may produce localized edema (lymphoedema). Any cause of tissue inflammation (infection, injury or ischemia) liberates vasodilator substances which cause vasodilatation and increase capillary permeability. Inflammatory edema is accompanied by the other features of inflammation - redness, tenderness and warmth - and is therefore painful. Increased capillary permeability occurs in acute allergic conditions (angioedema). The affected area may be red and itchy (pruritic) because of local release of histamine and other inflammatory mediators but, in contrast to inflammation, is not painful. Postural edema is due to failure of muscle movement and is common in the lower limbs of inactive patients. Finally, 'Idiopathic edema' occurs most commonly in women, typically varies with the menstrual cycle, and is characterised by marked diurnal variation in weight (more than 1.5 kg over 12 hours). This probably reflects variation in capillary permeability rather than extracellular fluid volume.

WHAT ARE THE CAUSES OF PHYSIOLOGIC EDEMA?
Physiologic edema is more common in advanced pregnancy, during hot weather, and in women who stand a lot.

WHAT ARE THE DRUGS THAT CAUSE EDEMA?
Steroids, NSAIDs, calcium channel antagonists, e.g. nifedipine, amlodipine

HOW TO APPROACH TO ANKLE OR LEG SWELLING?

S: Site O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Duration and Course) E: Exacerbating and precipitating factors S: Severity

SOCRATES

PRESENTATION: ANKLE OR LEG SWEELING

1. Where is the site of the swelling e.g. generalized, localized (ankle, scrotum, or face and hands)? Edema limited to one leg or to one or both arms is usually the result of venous and/or lymphatic obstruction. Edema resulting from hypoproteinemia and fluid overload is characteristically generalized.
2. What is the onset of the attack e.g. sudden or gradual?

3. What is the character of the swelling e.g. pruritic, painful, and/or dusky or red?
4. What are the relieving factors e.g. diuretics, sleeping, or resting?
5. What are the associated features? Local tenderness and warmth suggest inflammation. Local cyanosis may signify venous obstruction. In individuals who have had repeated episodes of prolonged edema, the skin over the involved areas may be thickened, indurated, and often red.
6. What is the timing of the swelling (duration and course)? Is it confined to the feet and ankles or does it extend to the shin, knee, thigh, or even the buttocks, genitalia, and anterior abdominal wall? Is there any evidence of abdominal swelling and ascites? Edema resulting from hypoproteinemia is especially evident in the very soft tissues of the eyelids (patient feels unable to fully open his eyes), face and hands (the ring on a finger fits more snugly than in the past) and tends to be most pronounced in the morning because of the recumbent posture assumed during the night. Edema associated with heart failure, by contrast, tends to be more extensive in the legs (patients feel shoes getting tight or complaining of difficulty in putting on shoes) and to be accentuated in the evening, a feature also determined largely by posture.
7. What are the exacerbating factors e.g. drugs, walking, sleeping, or menstruation?
8. What is the severity of the swelling?

EXAMPLES

During the early days of his illness course, the patient had noticed a swelling that initially involved the feet and ankles. This swelling had progressed gradually to ascend up and involve legs, thighs, and genitalia. The patient had noticed a particular difficulty opening his eye on waking in the morning and in wearing his ring; his face was puffier than in the past. In the last days of his illness course the patient claimed that all his body was swollen and that his weight started to increase. He had noticed no pain or bluish discoloration of his swelling as well as no sense of breathlessness.

What is the diagnosis?
The diagnosis is generalized edema due to hypoalbuminemia.

During the early days of his illness course, the patient had noticed a swelling that initially involved the feet and ankles. This swelling had progressed gradually to ascend up and involve legs, thighs, and genitalia; leg swelling is however became less noticeable in the morning when the patient started to slightly raised his legs over night. The patient had noticed a particular difficulty putting his feet in shoes in the evening. In the last days of his illness course the patient claimed that all his body parts was swollen and that his weight started to increase; he got some improvement when he started to use a medicine made him urinate more than usual. He had noticed no pain in his swelling but slight bluish discoloration of both legs as well as he started to experience sense of breathlessness when walking and at night when sleeping.

What is the diagnosis?
The diagnosis is generalized edema due to fluid overload e.g. cardiac failure.

The condition started three days after caesarian section operation when the patient had noticed left leg swelling extending from the ankle joint to the knee joint. The swelling was slightly discolored blue and was painful that rendered her unable to walk. The swelling was slightly improved when the patient raised her leg. The patient had noticed no change in the right leg and had not experienced shortness of breath or chest pain.

What is the diagnosis?
The diagnosis is localised edema due to deep venous thrombosis (DVT).

The condition started gradually over many days when the patient had noticed entire right leg swelling that was non painful, having normal color, and associated with heaviness like sensation in her abdomen especially in the inguinal region.

The patient had noticed no reddish discoloration of her right leg, no shortness of breath, and no chest pain but generally she felt weak and losing weight.

What is the diagnosis?
The diagnosis is localized edema (lymphoedema) due to lymphatic obstruction.

The condition started gradually over two days when the patient had noticed right leg swelling that was severely painful rendering her unable to walk, reddish in color, and associated with hotness. This event occurred after the patient walked bare-footed for a long distance. The patient had noticed no bluish discoloration of her right leg, no shortness of breath, and no chest pain but generally she felt weak and febrile.

What is the diagnosis?
The diagnosis is localized edema (inflammatory) due to cellulitis.

DYSPNEA

Dyspnea or breathlessness is an undue awareness of breathing. It is normal with strenuous physical exercise. It can be defined clinically as shortness of breath with some or all of the following:-

I. Abnormal increased inspiratory movement evident by:

- Dilation of alae nasi.
- Indrawing of suprasternal fossa, supraclavicular fossae, and intercostals spaces due to the use of accessory muscles (sternomastoids, platysma and pectoral muscles) of respiration to increase chest expansion during each inspiration. These muscles cause elevation of the shoulders with inspiration and aid respiration by increasing chest expansion. The use of accessory muscles is characteristic of patients with chronic obstructive pulmonary disease (COPD) and severe acute asthma.
- Abdominal paradox (Hoover's sign) that is indicative of diaphragmatic muscle fatigue.
- Tracheal descent during inspiration (tracheal tug).

II. Abnormal increased expiratory movement evident by:

- The patients assume upright or sitting position grasping a bed table or the back of the chair (Tripod position). This position stabilizes the shoulder girdles and helps the action of the accessory muscles.
- The patients exhale through their mouth with pursed lip and this is called (pursed lip breathing). Pursed-lip breathing provides a positive intra-airway pressure that is thought to decrease airway closure and air trapping and to aid gas exchange. Patients often discover this maneuver on their own or learn it through formal instruction by healthcare providers, although the exact benefit of this technique has not been established.

It is useful to understand the presentation of dyspnea. Dyspnea can be classified as acute (over a period of minutes to days) and chronic presentation (over months to years). Remember that there are five broad categories for causes of acute dyspnea as shown in BOX 1- 18. These are cardiac, respiratory, metabolic, psychological, and deconditioning (due to unaccustomized exercise) causes.

BOX 1 – 18. Some causes of dyspnea

	Acute dyspnea	Chronic dyspnea
Cardiovascular	Acute pulmonary edema	Chronic heart failure Myocardial ischemia
Respiratory	Acute exacerbation of asthma or COPD Pneumothorax Pneumonia Pulmonary embolism Acute respiratory distress syndrome Lobar collapse Laryngeal edema e.g. anaphylaxis	COPD Chronic asthma Interstitial lung disease Chronic pulmonary thromboembolism Bronchial carcinoma and lymphatic carcinomatosis Large pleural effusion (s)
Metabolic acidosis Kussmaul breathing	Diabetic ketoacidosis, uremia, overdose of salicylate, lactic acidosis, ethylene glycol poisoning	Sever anemia
Psychogenic	Hyperventilation (anxiety or panic-related)	
Other	Deconditioning in exercise	Obesity

Paroxysmal nocturnal dyspnea (PND) occurs in patient with left-sided heart failure is describes as a sudden breathlessness which wakes the patient from sleep choking or gasping for air. This happens because fluid shifts from

the interstitial tissues in the peripheries to the circulation within 1-2 hours of lying down in bed. Pulmonary edema may supervene, causing the patient to wake and sit upright, profoundly breathless. Patients may sit on the edge of the bed and open windows in an attempt to relieve their distress. It may be confused with asthma, which can also cause night-time symptoms, as it causes wheeze, but patients with heart failure often also produce frothy, bloodstained, sputum. Thus nocturnal dyspnea suggests congestive heart failure or asthma. Intermittent episodes of dyspnea are more likely to reflect episodes of arrhythmias, myocardial ischemia, bronchospasm, or pulmonary embolism. Orthopnea is defined as dyspnea in the supine posture (when patients lie flat) and occurs in congestive heart failure, respiratory failure, bilateral diaphragmatic paralysis, severe COPD, asthma, sleep apnea, or severe gastroesophageal reflux disease. Platypnoea is defined as dyspnea in the upright position with relief in the supine position. This type of dyspnea may suggest left atrial myxoma, hepatopulmonary syndrome, severe lung fibrosis, or after pneumonectomy. Trepopnea is dyspnea in the lateral decubitus position, as occurs with tumors of the main bronchi, unilateral pleural effusion, or after pneumonectomy.

HOW TO APPROACH TO DYSPNOEA?

S: Site-related (positional) O: Onset C: Course R: Relieving factors A: Associated factors
T: Timing (Day or night and duration) E: Exacerbating and precipitating factors S: Severity

SOCRATES

PRESENTATION: SHORTNESS OF BREATH

1. Is dyspnea induced by certain position e.g. orthopnea, trepopnea, or platypnoea? If the patient is orthopnoeic ask him or her about the numbers of pillows does he/she use.
2. What is the onset of dyspnea e.g. acute or chronic?
3. What is the course of dyspnea e.g. progressive or intermittent?
4. What are the relieving factors e.g. inhalation, oxygen, lying supine, or standing? Patients with acute pulmonary edema usually prefer to be upright, while those with pulmonary embolism are often more comfortable lying flat and may faint (syncope) if made to sit upright.
5. What are the associated symptoms e.g. cough, hemoptysis, and wheeze or chest pain?
6. What is the timing of dyspnea e.g. day time or night time, and duration? Breathlessness that wakes the patient from sleep is typical of asthma and left ventricular failure (paroxysmal nocturnal dyspnea). Patients with asthma typically wake between 3 and 5 a.m. and have associated wheezing (because at this time the steroid level is minimal inside the body). Breathlessness that is worst on waking in the morning is more typical of chronic obstructive pulmonary disease (COPD) and may improve after coughing up sputum.
7. What are the exacerbating and/or precipitating factors e.g. exercise, drugs, smoke, or stormy whether? Patients with exercise-induced asthma may notice that their breathlessness continues to worsen for 5-10 minutes after stopping activity. If you suspect asthma, ask about exposure to allergens, smoke, perfumes, fumes, cold air or drugs (aspirin or NSAIDs can trigger breathlessness). Common allergens are house dust mite (shaking bedding, hoovering), animals (cats, dogs, horses) and grass pollens and tree pollens. Breathlessness which improves at the weekend or on holiday suggests occupational asthma or extrinsic allergic alveolitis.
8. What is the severity of dyspnea? Ask about the effect of work, heavy exertion, walking up hills or walking with contemporaries. Severely breathless patients are dyspnoeic at rest and while walking around the house, getting washed, dressing, and eating or doing light housework. Use grading scales for breathlessness (BOX 1 – 19).

BOX 1 – 19. Severity of breathlessness: Medical Research Council (MRC) classification	
Grade 1	Breathless when hurrying on the level or walking up a slight hill
Grade 2	Breathless when walking with people of own age or on level ground
Grade 3	Has to stop because of breathlessness when walking on level ground at own pace

WHAT IS MEANT BY CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)?
COPD is the term used to describe slowly progressive airways obstruction, usually associated with smoking that is not reversible and is not due to another specific cause. COPD includes chronic bronchitis, small airways obstruction, and emphysema. Chronic bronchitis is a clinical diagnosis, defined as the presence of chronic cough and sputum production for at least 3 months of the year for at least 2 consecutive years in the absence of any other disease. Emphysema is defined as the enlargement of air spaces distal to the conducting airways, that is, respiratory bronchioles and alveoli, due to destruction of the walls of these air spaces.

EXAMPLES

The condition started suddenly two hours after sleeping at night as SOB that woke the patient from sleep too terrible, was so severe rendering him to stand up and open windows in attempt to get more air. The condition was associated with productive cough of frothy pink sputum. His condition had been slightly improved by standing and using of oxygenation in his home until few minutes where he asked for help and immediately rushed into the emergency unit of the nearest hospital. This attack was the third in this patient's course of illness. The first attack was managed in the hospital and the second attack eased by sitting and medicine intake in home.

What is the diagnosis?
The diagnosis is acute dyspnea (paroxysmal nocturnal dyspnea) with pulmonary edema.

The condition started instantaneously as SOB one hour after playing basketball. The attack was moderately severe made him unable to continue playing; a little pit improved with resting in a sitting position, and was associated with anterior chest pain. When the patient had noticed no complete improvement in his condition he asked his friends to be evacuated to the emergency unit of the nearest hospital. This attack was the second attack in this patient's life; the first one had been encountered one year ago whilst the patient was playing also and was managed in the emergency unit with the insertion of chest tube.

What is the diagnosis?
The diagnosis is acute dyspnea due to pneumothorax in patient with Marfan's syndrome. The patient was basket ball player as he was tall hence Marfan's syndrome.

The condition started gradually as an exertional SOB that was experienced after walking for about 500 meters in the early days of the patient's illness course. His condition had however deteriorated after few months and the SOB had been noticed with a lesser degree of exertion such as walking for 300 meters. One year after that he started to become short of breath with daily activity like for example bathing but activities like slow eating or talking slowly or resting did not result in breathlessness. His shortness of breath had been constantly of moderate severity in the last days, was mainly worst in the morning on waking, slightly improved with the use of inhalers, oxygen, and after coughing up sputum, and associated with small volume productive cough and wheeze. The patient had noticed three separated attacks of so severe breathlessness in the last year that was experienced even at rest and necessitating hospital admission.

What is the diagnosis?
The diagnosis is chronic dyspnea probably due to chronic obstructive pulmonary disease.

CLINICAL CLUE

HOW CAN YOU DISTINGUISH CARDIAC FROM PULMONARY DYSNPNEA? (POSTGRADUATE)
If a patient has evidence of both pulmonary and cardiac disease, a cardiopulmonary exercise test should be carried out to determine which system is responsible. If, at peak exercise, the patient demonstrates an increase in dead space or hypoxemia (oxygen saturation below 90%), or develops bronchospasm, the respiratory system is probably the cause of the problem. Alternatively, if the blood pressure becomes excessively high or drops during exercise, if the O_2 pulse falls, or if there are ischemic changes on the electrocardiogram, an abnormality of the cardiovascular system is likely the explanation for the breathing discomfort.

COUGH
Cough is the most frequent symptom of respiratory disease (BOX 1 – 20). It is caused by stimulation of sensory nerves in the mucosa of the pharynx, larynx, trachea and bronchi.

BOX 1 – 20. Causes and characters of cough

Origin	Common causes	Clinical features
Pharynx	Post-nasal drip	History of chronic rhinitis
Larynx	Laryngitis, tumour,	Voice or swallowing altered, harsh or painful cough
	Whooping cough, croup	Paroxysms of cough, often associated with stridor
Trachea	Tracheitis	Raw retrosternal pain with cough
Bronchi	Bronchitis (acute) and COPD	Dry or productive, worse in mornings
	Asthma	Usually dry, worse at night
	Bronchial carcinoma	Persistent (often with hemoptysis)
Lung parenchyma	Tuberculosis	Productive, often with hemoptysis
	Pneumonia	Dry initially, productive later
	Bronchiectasis	Productive, changes in posture induce sputum production
	Pulmonary edema	Often at night (may be productive of pink, frothy sputum)
	Interstitial fibrosis	Dry, irritant and distressing

The most common causes of cough can be categorized according to the duration of the cough. Acute cough (<3 weeks) is most often due to upper respiratory infection (common cold, acute bacterial sinusitis, and pertussis), but more serious disorders, such as pneumonia, pulmonary embolus, and congestive heart failure, can also present in this fashion. Subacute cough (between 3 and 8 weeks) is commonly post-infectious, resulting from persistent airway inflammation and/or postnasal drip following viral infection, pertussis, or infection with Mycoplasma or Chlamydia. Chronic cough (> 8 weeks) in a smoker raises the possibilities of chronic obstructive lung disease or bronchogenic carcinoma. Non smoker patients with chronic cough present more of a diagnostic challenge, especially those individuals with a normal examination, chest X-ray and lung function studies. In this context, most cough can be explained by one or more of the following (mnemonic: ABCDE2)

- Angiotensin-converting enzyme (ACE) inhibitors intake. Ten to fifteen per cent of patients (particularly women) taking this drug develop chronic cough. The cough can start from one week up to a year after starting the treatment. The mechanism of ACE inhibitor cough may involve sensitization of sensory nerve endings due to accumulation of bradykinin.
- Bordetella pertussis infection (whooping cough) in adults can also result in protracted cough and should always be suspected in those in close contact with children.
- Cough – variant asthma (where cough may be the principal or exclusive clinical manifestation).
- Post – nasal Drip secondary to nasal or sinus disease.
- Gastro – Esophageal reflux with aspiration.
- Eosinophilic bronchitis.

WHAT IS THE REASON OF BOVINE COUGH?
The explosive quality of a normal cough is lost in patients with respiratory muscle paralysis or vocal cord palsy. Paralysis of a single vocal cord gives rise to a prolonged, low-pitched, inefficient 'bovine' cough accompanied by hoarseness.

HOW TO APPROACH TO COUGH?

SOCRATES

S: Site-related (positional) O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Day or night and duration) E: Exacerbating and precipitating factors S: Severity

PRESENTATION: COUGH

1. Does the cough induce or increase in frequency by certain positions?
2. What is the onset of the cough e.g. acute, subacute, or chronic?
3. What is the character of the cough e.g. dry or productive, painful or painless? Does it occur in bouts suggestive of whooping cough?
4. What are the relieving factors e.g. drugs?
5. What is the associated symptoms e.g. nasal discharge, fever, wheeze, or hoarseness?

6. What is the timing of the cough e.g. day time or night time, and duration?
7. What are the exacerbating and precipitating factors e.g. cold, dust or pollen?
8. What is the severity of the cough? How frequent is it?

SPUTUM PRODUCTION

Sputum production is common in patient with acute or chronic cough and its nature and appearance can provide valuable clues to the etiology (BOX 1 – 21).

BOX 1 – 21. Character of sputum

Type	Appearance	Cause
Serous	Frothy, pink	Acute pulmonary oedema
	Clear, watery	Alveolar cell cancer (bronchorrhea)
Mucoid	Clear, grey	Chronic bronchitis/COPD
	White, viscid	Asthma
Purulent	Yellow	Acute bronchopulmonary infection (live neutrophils)
		Asthma (eosinophils)
	Green	Longer-standing infection (dead neutrophils) • Pneumonia • Bronchiectasis • Cystic fibrosis • Lung abscess
Rusty	Rusty red	Pneumococcal pneumonia

CLINICAL CLUE

Bronchorrhea means expectoration of thin serous fluid of more than 100 cc daily and occurs in 20% of patients with alveolar cell carcinoma. Bronchorrhea may also be noticed in patients with allergic bronchopulmonary aspergillosis and in organophosphorus compound poisening.

HOW TO APPROACH TO SPUTUM?

AMOUNT/ASSOCIATION BLOOD/POSITION
COLOR/CHARACTER DURATION/DAY OR NIGHT TIME

A/A B/P C/C D/D

PRESENTATION IS RELATED TO COUGH

1. What is the AMOUNT? Is it a small (a teaspoonful) or large (a teacupful) amount? Bronchiectasis causes large volumes of purulent sputum to be coughed up, varying with posture. Large volumes of watery sputum with a pink tinge in an acutely breathless patient suggest pulmonary edema, but occurring over weeks (bronchorrhoea) suggests alveolar cell cancer. Sudden production of large amounts of purulent sputum on a single occasion suggests rupture of a lung abscess or empyema into the bronchial tree.
2. What are the ASSOCIATED symptoms e.g. dyspnea, fever, weight loss, or night sweat?
3. Is it ever BLOOD – stained?
4. At what POSITION is sputum most produced? In patients with bronchiectasis purulent sputum is varying with posture.
5. What are the COLOR, TASTE, and SMELL of the sputum? Yellow sputum occurs in acute lower respiratory tract infection (due to the presence of live neutrophils) and in asthma (due to the presence eosinophils) while green sputum (due to dead neutrophils) indicates longer standing infection; dead neutrophils release the green-pigmented enzyme, verdoperoxidase. In early pneumococcal pneumonia sputum may be a rusty red color because pneumonic inflammation causes lysis of red cells. Chronic bronchitis and COPD cause clear or 'mucoid' sputum if there is no active infection while the first sputum produced in the morning by a patient with COPD may be green because of nocturnal stagnation of neutrophils. 'Foul' or 'vile'-tasting or -smelling sputum suggests

anaerobic bacterial infection, and occurs in bronchiectasis, lung abscess and empyema. In bronchiectasis a change of sputum taste may indicate an infective exacerbation.

6. What is the CHARACTER of the sputum e.g. serous, mucoid, mucopurulent or purulent (FIGURE 1 – 3)?
7. What is the DURATION of the attack?
8. At which TIME of the day does sputum is produced?

CLINICAL CLUE

In asthma and allergic bronchopulmonary aspergillosis viscid secretions can accumulate in airways and be coughed up as 'worm-like' structures that are casts of the bronchi. Other solid matter sometimes coughed up includes necrotic tumour and inhaled foreign bodies, e.g. food, teeth and tablets.

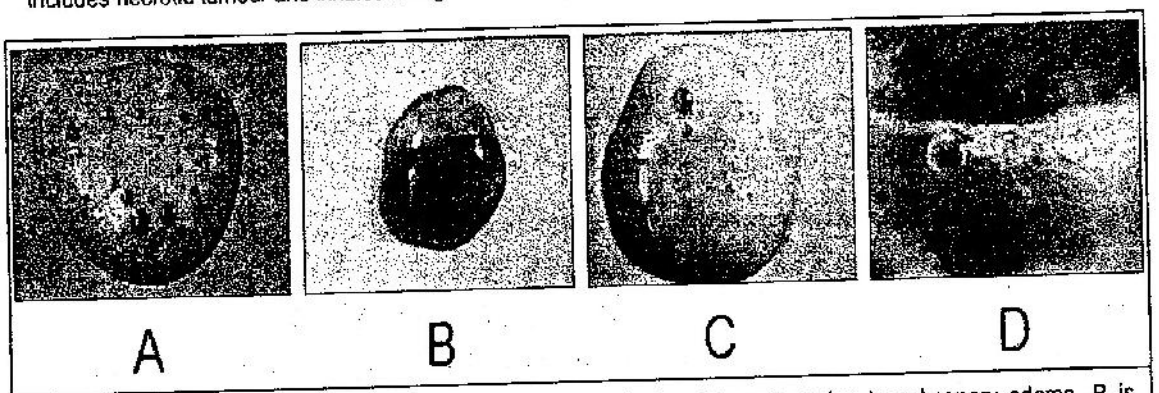


FIGURE 1 – 3. Some samples of sputum. A is a serous frothy pink sputum due to pulmonary edema. B is mucopurulent sputum due to bronchial or pneumonic infection. C is purulent sputum due to bronchial or pneumonic infection. D is blood stained-stained sputum due to cancer, tuberculosis, bronchiectasis, or pulmonary embolism.

HOW CAN YOU DISTINGUISH SALIVA FROM SPUTUM?
Because sputum consists mainly of secretions from the tracheobronchial tree rather than the upper airway, the finding of alveolar macrophages and other inflammatory cells is consistent with a lower respiratory tract origin of the sample i.e. true sputum, whereas the presence of squamous epithelial cells in a "sputum" sample indicates contamination by secretions from the upper airways i.e. presence of saliva. To be adequate for culture, a sputum sample must have >25 neutrophils and <10 squamous epithelial cells per low-power field.

EXAMPLES

The condition started five days ago when the patient had noticed burning and tickling sensation in the nose, blocked nose, and watery yellow – green nasal discharge that persisted for two days. In the third day the patient had complained from short painful dry cough with retrosternal chest pain and low grade intermittent fever. In the fifth day the patient started to have productive cough of teaspoonful amount, not blood stained, yellow to green in color, and purulent. No particular position had been noticed to induce or increase the cough that was so frequent, present all the time, slightly eased with cough syrup, and became associated with mild SOB and higher grade fever with shaking chills.

What is the diagnosis?
The diagnosis is acute coryza (common cold) complicated by pneumonia.

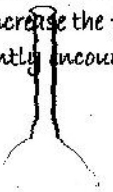
The condition started two months ago as low grade intermittent fever, night sweat, and cough. No particular position had been noticed to induce or increase the cough that over the days of the first month was dry, so frequent, and present all the time; slightly eased with cough syrup, and associated with SOB, low grade fever, and weight loss. The cough over the second month became productive of teacupful amount; yellow in the start then became green in color with blood streaking and purulent.

What is the diagnosis?
The diagnosis is post primary tuberculosis.

The condition started two months ago as low grade intermittent fever, night sweat, weight loss with decreasing appetite and cough. Sleeping on the left side had induced or increased the frequency of the cough that was productive, so frequent about twenty times daily, and present all over the day but in particular at morning time; slightly eased with cough syrup, and associated with shortness of breath. The sputum was yellow in the start of the illness course then became green and purulent with blood streaking, of copious (teacupful) amount, and foul in smell and taste. Sleeping on the left side had increased the amount of sputum produced.

What is the diagnosis?
The diagnosis is bronchiectasis.

The condition started gradually one year ago as an exertional shortness of breath and cough. No particular position had been noticed to induce or increase the frequency of cough that was present all over this time, so irritant, distressing, and frequently encountered all over the day. Cough syrup had slightly eased the cough.



What is the diagnosis?
The diagnosis is pulmonary fibrosis.

HEMOPTYSIS

Coughing up blood, irrespective of the amount, is an alarming symptom and nearly always bring the patient to the doctor. A clear history should be taken to establish that is a true hemoptysis and not hematemesis or epistaxis (nosebleed). A history of repeated small hemoptysis or blood streaking of sputum is highly suggestive of bronchial carcinoma. Chronic fever and weight loss may suggest tuberculosis. Pneumococcal pneumonia is often the cause of rusty-colored sputum but can also cause frank hemoptysis. Intracavitary aspergilloma can cause bronchial hemorrhage. Pulmonary thromboembolism is a common cause of hemoptysis and should always be considered.
Many episodes of hemoptysis are unexplained, even after full investigation, and are likely to be caused by simple bronchial infection. The causes of hemoptysis are provided in BOX 1 – 22.

BOX 1 – 22. Causes of hemoptysis

Bronchial disease	Parenchymal disease	Lung vascular disease	Cardiovascular disease	Blood disorder
Acute bronchitis Bronchial adenoma or carcinoma Bronchiectasis Foreign body	Tuberculosis Lung abscess Pneumonia Trauma Parasite (e.g. hydatid disease)	Pulmonary infarction Pulmonary renal syndrome: • Goodpasture's syndrome. • Wegener's granulomatosis. • Microscopic polyangiitis. • Churg-Strauss syndrome. • Cryoglobulinaemia	Acute left ventricular failure Mitral stenosis Aortic aneurysm	Leukemia Hemophilia Anticoagulation

While precise epidemiologic data are lacking, the most common etiology of hemoptysis is viral or bacterial bronchitis. Worldwide, the most common cause of hemoptysis is tuberculous infection presumably owing to the high prevalence of the disease and its predilection for cavity formation.

CLINICAL CLUE

Because blood originating from the nasopharynx or the gastrointestinal tract (pseudohemoptysis) can mimic blood coming from the lower respiratory tract, it is important to determine initially that the blood is not coming from one of these alternative sites. Clues that the blood is originating from the gastrointestinal tract include a dark red appearance and an acidic pH, in contrast to the typical bright red appearance and alkaline pH of true hemoptysis.

HOW TO APPROACH TO HEMOPTYSIS?

P: Pseudohemoptysis or hemoptysis A: Amount N: Nature of blood
D: Duration of the attack A: Associated symptoms

PANDA

PRESENTATION: COUGHING UP BLOOD

1. Is there any blood in sputum as fear often leads a patient not to mention it?
2. Is it true hemoptysis or Pseudohemoptysis?
3. What is the **A**mount of hemoptysis? Large-volume hemoptysis, referred to as massive hemoptysis, is variably defined as hemoptysis of greater than 200–600 cc in 24 h. Massive hemoptysis should be considered a medical emergency.
4. What is the **N**ature of hemoptysis i.e. frothy pink sputum, streaks of blood, or clear blood (bright red or dark red)?
5. What is the **D**uration of the attack?
6. What are the **A**ssociated symptoms?

EXAMPLES

The condition started rapidly as burning and tickling sensation in the nose, blocked nose, and watery yellow - green nasal discharge that persisted for one day. In the next day the patient had complained from irritating non productive painful cough that was associated with wheeze, shortness of breath, and low grade intermittent fever. The cough after that started to produce scanty amount of mucoid sputum. After that the cough started to be more copiers in amount and of mucopurulent blood-stained sputum over the next three days. The blood was mixed with the sputum, four times daily for the next two days, little in amount, and bright red in color. The patient was sure that the blood is the result of coughing and not vomiting or nose bleeding. The patient after that spontaneously recovered.

What is the diagnosis?
The diagnosis is acute coryza (common cold) complicated by acute bronchitis.

The condition started three months ago as low grade intermittent fever, night sweat, and extreme weight loss with reduced appetite and cough. No particular position had been noticed to induce or increase the cough that was dry initially, frequent, present all the time, slightly eased with cough syrup, and associated with shortness of breath. The cough then became productive of scanty yellowish sputum with streaks of blood on the sputum over the preceding month. The sputum over the last two days started to produce massive blood expectoration with sputum; two cupful sputum in a day.

What is the diagnosis?
The diagnosis is bronchial carcinoma.

VOMITING

Nausea is the subjective feeling of a need to vomit. Vomiting is the oral expulsion of gastrointestinal contents resulting from contractions of gut and thoracoabdominal wall musculature. Retching is a strong involuntary effort to vomit. Vomiting is contrasted with regurgitation, the effortless passage of gastric contents into the mouth. Rumination is repeated regurgitation of stomach contents, which may be rechewed and reswallowed. In contrast to vomiting, these phenomena often exhibit volitional control. Some causes of vomiting are provided in BOX 1 – 23.

BOX 1 – 23. Some causes of vomiting

Infections	CNS disorders	Gastroduodenal disease
Gastroenteritis	Migraine	Chronic peptic disease
Hepatitis	Raised intracranial pressure	Gastroparesis e.g. diabetes, drugs
Urinary tract infection	Meningitis	Gastric cancer
Drugs	**Acute abdominal disorders**	**Psychiatric illness**
Cytotoxic drugs	Appendicitis	Anorexia nervosa
Antibiotics	Cholecystitis	Bulimia nervosa
Digoxin	Pancreatitis	Depression
NSAIDs and opiates	Intestinal obstruction	
Metabolic	**Labyrinthine disease**	**Others**
Diabetic ketoacidosis	Motion sickness	Any severe pain e.g. myocardial infarction
Uremia	Labyrinthitis	Psychogenic
Addison's disease	Malignancy	Alcohol

HOW TO APPROACH TO VOMITING?

S: Severity O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Course and Duration) E: Exacerbating and precipitating factors S: Severity

SOCRATES

PRESENTATION: VOMITING

1. What is the severity of vomiting? How frequent is the vomiting?
2. What is the onset of the vomiting? Drugs, toxins, and gastrointestinal infections commonly cause acute symptoms, whereas established illnesses evoke chronic complaints. Chronic nausea and/or vomiting with no other abdominal symptoms is usually due to a psychological cause.
3. What is the character of vomiting e.g. contents, color, projectile or effortless, and the amount of each vomitus? Emesis of undigested food is consistent with a Zenker's diverticulum or achalasia. Blood in vomiting (hematemesis) raises suspicion of an ulcer, malignancy, or Mallory-Weiss tear whereas bilious vomiting excludes gastric obstruction. Feculent emesis is noted with distal intestinal or colonic obstruction or the presence of a gastrocolic fistula. Projectile vomiting can be due to raised intracranial pressure or due to gastric-outflow obstruction. Large volumes of vomit suggest intestinal obstruction.
4. What are the relieving factors e.g. drugs?
5. What are the associated symptoms with vomiting e.g. abdominal pain, fever, vertigo, or headache? Does the vomiting preceded by nausea, retching, or giddiness? Many gastrointestinal conditions are associated with vomiting, but nausea and vomiting without pain is frequently non-gastrointestinal in origin. Relief of abdominal pain by emesis characterizes small-bowel obstruction whereas vomiting has no effect on pancreatitis or cholecystitis pain. Fevers suggest inflammation; an intracranial source is considered if there are headaches or visual field changes; and vertigo or tinnitus indicates labyrinthine disease.
6. What is the timing of vomiting e.g. duration and course? Does it occur at the early morning time? Early-morning vomiting is due to psychological cause, pregnancy, alcohol dependence, metabolic disorders, and in case of increased intracranial pressure.
7. What are the exacerbating and the precipitating factors e.g. drugs or foods?

DYSPHAGIA

Dysphagia is defined as a difficulty in swallowing. Aphagia signifies complete esophageal obstruction which is usually due to bolus impaction and represents a medical emergency. Odynophagia means painful swallowing and usually results from candidal, herpes, pill-induced or reflux-induced esophagitis. Globus pharyngeus is the sensation of a lump lodged in the throat. However, no difficulty is encountered when swallowing is performed. Phagophobia, meaning fear of swallowing, and refusal to swallow may occur in hysteria, rabies, tetanus, and pharyngeal paralysis due to fear of aspiration. The upper esophageal sphincter and the muscle of the proximal one-third of the esophagus

are skeletal muscle under voluntary control. A transition from skeletal to smooth muscle occurs in the mid-esophagus. In the distal one-third of the esophagus, the muscle is smooth muscle that is under involuntary control. Dysphagia can be classified according to the site of swallowing difficulty into oropharyngeal and esophageal causes. When dysphagia arises from disease involving the region of skeletal muscle, it is called oropharyngeal dysphagia. Oropharyngeal dysphagia is characteristically accompanied by failure to initiate swallowing to transport a bolus from the mouth to the esophagus, by nasal regurgitation because of lack of occlusion of the nasal air passages by the soft palate, and by coughing with swallowing because of aspiration resulting from failure to elevate the larynx and lower the epiglottis. Oropharyngeal dysphagia results from neurological dysfunction (bulbar or pseudobulbar palsy and myasthenia gravis). Drooling, dysarthria, hoarseness and cranial nerve or other neurological signs may be present. When dysphagia arises from disease involving the region of smooth muscle, the impairment in bolus transport is perceived substernally and called esophageal dysphagia. Furthermore, esophageal dysphagia (FIGURE 1 – 4) can be readily separated into (1) diseases that cause dysphagia for solids only, indicative of a lumen-narrowing (mechanical) lesion such as lower esophageal rings, peptic stricture, or cancer (swallowing of liquid is normal until strictures become extreme) and (2) diseases that cause dysphagia for liquids as well as solids, indicative of motor dysfunction such as diffuse esophageal spasm, progressive systemic sclerosis (scleroderma), and achalasia.

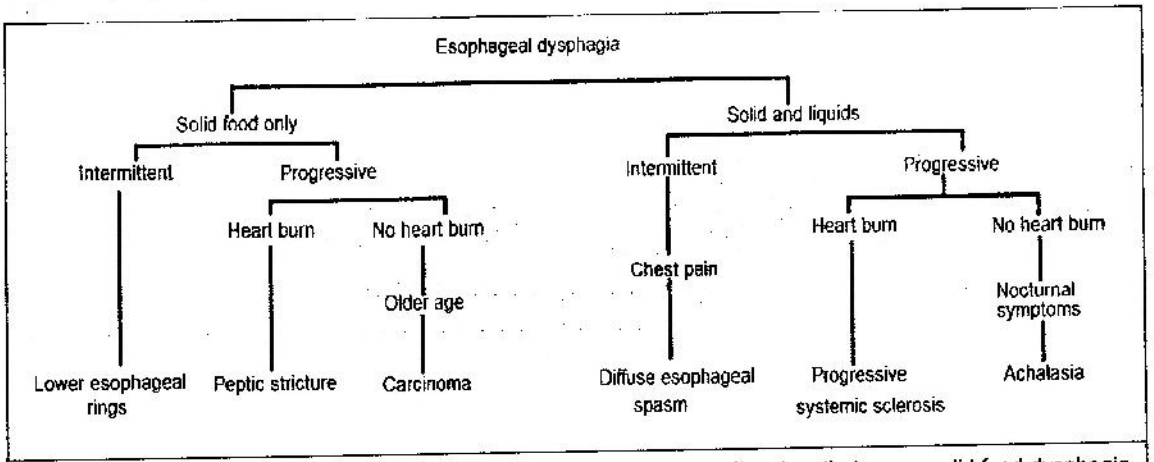


FIGURE 1 – 4. Approach to esophageal dysphagia. Remember that the disorders that cause solid food dysphagia usually causing mechanical dysphagia due to narrow lumen while these disorders that cause solid and liquid dysphagia usually causing motor dysphagia.

Esophageal rings are common cause of dysphagia. Esophageal ring, called Schatzki ring, is a thin, web-like constriction located at or near the border of the lower esophageal sphincter. Esophageal webs are thin, fragile membranes that occur in the upper and midportion of the esophagus. The combination of dysphagia due to esophageal webs and iron-deficiency anemia in middle-aged women constitutes Plummer-Vinson syndrome. Rings or webs may cause intermittent dysphagia to solids. Either may be congenital or could be the result of inflammatory response e.g. GERD or eosinophilic esophagitis. Symptomatic rings and webs are easily treated by dilatation.
Peptic strictures develop as a consequence of long-standing esophagitis due to GERD or peptic ulcer. Most patients are elderly and present with dysphagia which is worse for solids than for liquids. A history of heartburn is common but not invariable; many elderly patients presenting with strictures have no preceding heartburn.

CLINICAL CLUE

In an adult, the esophageal lumen can distend up to 4 cm in diameter. When the esophagus cannot dilate beyond 2.5 cm in diameter, dysphagia to normal solid food can occur. It is always present when the esophagus cannot distend beyond 1.3 cm.

HOW TO APPROACH TO DYSPHAGIA?

SOCRATES

S: Site O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Course and Duration) E: Exacerbating and precipitating factors S: Severity

PRESENTATION: DIFFICULTY IN SWALLOWING

1. Is there difficulty in swallowing, painful swallowing, fear of swallowing, or globus sensation?
2. If the complain is dysphagia where is the site of swallowing difficulty i.e. does the patient have difficulty initiating swallowing (oropharyngeal dysphagia) or food sticking after swallowing (esophageal dysphagia)? Is there any nasal regurgitation, choking, or aspiration? Nasal regurgitation and aspiration with swallowing are hallmarks of oropharyngeal dysphagia. Bronchial aspiration unrelated to swallowing may be due to achalasia, Zenker's (pharyngeal) diverticulum, or gastroesophageal reflux.
3. If the complain is esophageal dysphagia where is the site at which the foods appear to stick? The site of dysphagia described by the patient helps to determine the site of esophageal obstruction; the lesion is at or below the perceived location of dysphagia.

CLINICAL CLUE

The rationale behind performing a swallow test in patients with stroke is to discover whether they have oropharyngeal dysphagia.

4. What is the onset of the attack e.g. acute or chronic?
5. What is the character of swallowing difficulty? Is it intermittent or progressive? Does the difficulty appear most to solid food or liquid and solid foods? Difficulty only with solids implies mechanical dysphagia with a lumen that is not severely narrowed. In advanced obstruction, dysphagia occurs with liquids as well as solids. In contrast, motor dysphagia due to achalasia and diffuse esophageal spasm is equally affected by solids and liquids from the very onset.
6. What are the relieving factors e.g. water drinking or standing?
7. What are the associated symptoms e.g. hoarseness, hiccups, wheezing, weight loss, heart burn, or chest pain? Association of laryngeal symptoms and dysphagia occurs in various neuromuscular disorders. When hoarseness precedes dysphagia, the primary lesion is usually in the larynx; hoarseness following dysphagia may suggest involvement of the recurrent laryngeal nerve by extension of esophageal carcinoma. Sometimes hoarseness may be due to laryngitis secondary to gastroesophageal reflux. Hiccups may rarely occur with a lesion in the distal portion of the esophagus. Unilateral wheezing with dysphagia may indicate a mediastinal mass involving the esophagus and a large bronchus. Severe weight loss that is out of proportion to the degree of dysphagia is highly suggestive of carcinoma. Chest pain with dysphagia occurs in diffuse esophageal spasm and related motor disorders. A prolonged history of heartburn and reflux preceding dysphagia indicates peptic stricture.
8. What is the timing of swallowing difficulty e.g. duration and course?
9. What are the precipitating and the relieving factors? Patients with scleroderma have dysphagia to solids that is unrelated to posture and to liquids while recumbent but not upright.
10. What is the severity of swallowing difficulty? Determine the impact of dysphagia upon the patient.

EXAMPLES

The condition started gradually over the preceding month as an intermittent difficulty in initiating swallowing. This difficulty became most noticeable after prolonged chewing as in meat chewing, particularly experienced in the later hours of the day, improved by rest and sleep, and associated with aspiration, chocking, disturbed speech, and muscle weakness. During the early hours of the day, she had noticed that she was completely normal.

What is the diagnosis?
The diagnosis is oropharyngeal dysphagia due to myasthenia gravis.

The condition started gradually over the preceding six months as a difficulty in swallowing. This difficulty was intermittent in nature, most noticeable for solid food only where the food became sticking after swallowing at the mid sternum, eased by fluid intake, and not associated with chest pain, aspiration, or chocking. Of note the patient had no prior history of heart burn, or concomitant complain of muscle weakness.

What is the diagnosis?
The diagnosis is esophageal dysphagia due to lower esophageal rings.

The condition started gradually over the preceding six months as a difficulty in swallowing. This difficulty was progressive in nature noticed in early months of the illness course for solid food only Where the food became sticking after swallowing at the mid sternum, eased by fluid intake, and not associated with aspiration or chocking during eating. The patient then developed difficulty to both solid and liquid diet. Of note the patient had prolonged prior history of heart burn, regurgitation of sour material into the mouth, and occasional chest pain.

What is the diagnosis?
The diagnosis is esophageal dysphagia due to peptic strictures.

The condition started gradually over the preceding ten months as a difficulty in swallowing. This difficulty was progressive in nature noticed for solid and liquid diet from the start where the food became sticking after swallowing at the mid sternum, eased somewhat by standing and moving around during eating, worsened by emotional stress and hurried eating, and associated with difficult belching, night cough, pulmonary aspiration, and weigh loss. The swallowing was not associated with chocking. Of note the patient had no prior history of heart burn or concomitant complain of muscle weakness.

What is the diagnosis?
The diagnosis is esophageal dysphagia due to achalasia.

GASTROINTESTINAL BLEEDING

Bleeding from the gastrointestinal tract is divided into upper gastrointestinal tract bleeding (proximal to the ligament of Treitz) and manifest clinically as hematemesis, melena, and some times hematochezia and lower gastrointestinal tract bleeding (distal to the ligament of Treitz) and manifest clinically as hematochezia and sometimes as melena. Ligament of Treitz is a suspensory ligament where fourth portion of the duodenum transitions to jejunum. Hematemesis is defined clinically as vomiting up blood that may be red with clots when bleeding is profuse, or black ('coffee grounds') when less severe. Melena is the term used to describe the passage of black, tarry stools containing altered blood; this is usually due to bleeding from the upper gastrointestinal tract, although hemorrhage from the right side of the colon is occasionally responsible. The characteristic appearance is the result of the action of digestive enzymes and of bacteria upon hemoglobin. Melena indicates that blood has been present in the gastrointestinal tract for at least 14 hour. Thus, the more proximal the bleeding site, the more likely melena will occur. Severe acute upper gastrointestinal bleeding can sometimes cause hematochezia. Hematochezia is defined as the passage of red blood in the stools. Hematochezia usually represents a lower gastrointestinal source of bleeding, although an upper gastrointestinal lesion may bleed so briskly that blood does not remain in the bowel long enough for melena to develop. When hematochezia is the presenting symptom of upper gastrointestinal bleeding, it is associated with hemodynamic instability and dropping hemoglobin. Occult gastrointestinal bleeding is defined as the detection of asymptomatic blood loss from the gastrointestinal tract, generally by routine fecal occult blood testing or the presence of iron deficiency anemia. Obscure gastrointestinal bleeding is defined as bleeding of unknown origin that persists or recurs after a normal initial endoscopic evaluation of both the upper and lower gastrointestinal tracts. Peptic ulcers are the most common cause of upper gastrointestinal bleeding (BOX 1 – 24), accounting for 50% of cases. Mallory-Weiss tears account for 5-15% of cases. The proportion of patients bleeding from varices varies widely from 5-30%, depending on the population.

HOW CAN YOU CLINICALLY DIFFERENTIATE UPPER FROM LOWER GASTROINTESTINAL BLEEDING?
Hematemesis is indicative of upper gastrointestinal bleeding.
Melena is mostly indicative of upper gastrointestinal bleeding.
Hematochezia is mostly indicative of lower gastrointestinal bleeding.
Hyperactive bowel sounds is indicative of upper gastrointestinal bleeding.
Elevated blood urea nitrogen level (due to volume depletion and blood proteins absorbed in the small intestine) is indicative of upper gastrointestinal bleeding.

BOX 1 – 24. Causes of upper gastrointestinal bleeding

Esophagitis	Mallory – Weiss tear	Gastric erosion	Vascular malformation
Esophageal varices	Esophageal or gastric tumor	Peptic ulcer	Aorto-duodenal fistula

Repeated vomiting, retching, or vigorous coughing can cause nontransmural tear at the gastroesophageal junction called Mallory-Weiss tear. Most patients present with hematemesis. Bleeding usually abates spontaneously, but protracted bleeding may occur. Aortoduodenal fistula is most commonly occur after surgery for an aortic aneurysm and may be related to infection of the graft and can result in gastrointestinal bleeding. The term 'peptic ulcer' refers to an ulcer in the lower oesophagus, stomach or duodenum, in the jejunum after surgical anastomosis to the stomach or, rarely, in the ileum adjacent to a Meckel's diverticulum. Gastric ulcers penetrate the muscularis mucosae while gastric erosions do not.

CLINICAL CLUE

FOR POSTGRADUATE STUDENTS

Characteristics of an ulcer at endoscopy provide important prognostic information. Poor endoscopic findings include active bleeding vessel, non-bleeding visible vessel, and adherent clot. Better findings include flat pigmented spot and clean base ulcer.

Surgical consultation in the management of acute gastrointestinal bleeding is indicated when bleeding is failed to respond to endoscopic treatment, in patients who rebleed, in patients who require large numbers of transfusions, and for large ulcers of the lesser curvature of the stomach or posterior wall of the duodenum.

Measurement of the heart rate and blood pressure is the best way to assess a patient with gastrointestinal bleeding. Clinically significant gastrointestinal bleeding leads to postural hypotension and tachycardia and finally recumbent hypotension. The hemoglobin does not fall immediately with acute gastrointestinal bleeding, due to proportionate reductions in plasma and red cell volumes (i.e., "people bleed whole blood"). Thus, hemoglobin may be normal or only minimally decreased at the initial presentation of a severe bleeding episode. As extravascular fluid enters the vascular space to restore volume, the hemoglobin falls, but this process may take up to 72 h. Patients with slow, chronic gastrointestinal bleeding may have very low hemoglobin values despite normal blood pressure and heart rate.

CLINICAL CLUE

Following repeated vomiting or retching, non-transmural tear at the gastroesophageal junction called Mallory-Weiss tear while transmural tear is called Boerhaave's syndrome. In the latter, severe chest pain and shock occur as oesophago-gastric contents enter the mediastinum and thoracic cavity. Subcutaneous emphysema, pleural effusions and pneumothorax develop.

Lower gastrointestinal bleeding may be due to hemorrhage from the small bowel, colon or anal canal. It is useful to distinguish those patients who present with profuse, acute bleeding from those who present with chronic or subacute bleeding of lesser severity (BOX 1 – 25).

BOX 1 – 25. Causes of lower gastrointestinal bleeding — ALI'S CAR

Severe/acute	Moderate, chronic/subacute	
Diverticular disease	Anal disease e.g. fissure or hemorrhoids	Carcinoma
Angiodysplasia	Large polyps	Angiodysplasia
Ischemia	Inflammatory bowel disease	Radiation enteritis
Meckel's diverticulum	Solitary rectal ulcer	

Angiodysplasias are painless dilated, thin-walled, red submucosal vessels that radiate from a central feeding vessel. Affected patients may present with lower gastrointestinal bleeding. Some suggests association with chronic renal disease, von Willebrand disease, and aortic stenosis (Heyde's syndrome).

HOW TO APPROACH TO HEMATEMESIS AND MELENA?

SOCRATES

S: Site O: Onset C: Character R: Relieving factors A: Associated factors

T: Timing (Course and Duration) E: Exacerbating and precipitating factors S: Severity

PRESENTATION OF HEMATEMESIS: VOMITING OF BLOOD

1. Does it genuine hematemesis or hemoptysis? It is important to ensure the site from which the blood is coming. Thus it is important to ensure if vomiting of blood is reported, this is not the result of bleeding from a tooth socket or the nose, or coughing up of blood.
2. What is the onset i.e. does the attack comprise a sudden or gradual process?
3. What is the character e.g. fresh blood or coffee ground-stained fluid, in the first vomitus or subsequent vomitus? Hematemesis can be fresh and red when severe or degraded by gastric pepsin and becomes dark brown in color and resembles coffee grounds when less severe. If the source of bleeding is above the gastro-oesophageal sphincter, e.g. from oesophageal varices, fresh blood may well up in the mouth, as well as being actively vomited. With a lower oesophageal mucosal tear due to the trauma of retching (Mallory-Weiss syndrome) the patient vomits forcefully several times and fresh blood only appears after the initial vomit.
4. What are the relieving factors e.g. drugs?
5. What are the associated features e.g. black tarry stools, weight loss, jaundice?
6. What is the timing of hematemesis (course and duration)?
7. What are the precipitating and the exacerbating factors e.g. alcohol, corticosteroid or NSAID therapy?
8. What is the severity of the attack (amount and the frequency)?

CLINICAL CLUE

FOR POSTGRADUATE STUDENTS

Intestinal ischemia is categorized into three types. Acute mesenteric ischemia (acute small bowel ischemia) is a life-threatening condition that is characterised by the classic teaching of "abdominal pain out of proportion to examination" because the physical examination early in the course is unremarkable. Most patients with this condition are older than 50 years. Although occult blood-positive stool is common, overt bleeding is rare. Late signs and symptoms include vomiting, fever, hematemesis, obstruction, back pain, and shock. Chronic mesenteric ischemia, also called intestinal angina or intestinal claudication, is characterized by severe, dull postprandial mid- or upper- abdominal pain that usually begins 30 minutes after eating and may last up to 3 hours; fear of eating typifies the disorder and invariably leads to weight loss. This type of ischemia results from atherosclerotic stenosis affecting at least two of the coeliac axis, superior mesenteric artery, and inferior mesenteric artery. Acute colonic ischemia, also called ischemic colitis, is much more common than acute or chronic mesenteric ischemia. The most commonly affected sites of colonic ischemia are the splenic flexure and the descending colon. Most patients with colonic ischemia are older than 60 years and usually present with left-lower quadrant pain, urgent defecation, and red or maroon rectal bleeding that does not require transfusion.

PRESENTATION OF MELENA: PASSAGE OF BLACK TARRY STOOLS

1. Where is the site of bleeding i.e. upper or lower gastrointestinal bleeding? Fresh bleeding per rectum indicates hematochezia and in the absence of hemodynamic instability the likely source of bleeding is lower gastrointestinal region while black tarry stools indicates melena and the likely source of bleeding is upper gastrointestinal region.
2. What is the onset of the attack e.g. sudden or gradual?
3. What is the character of the stools (tarry and shiny black or matt black stool)? Is blood passed with it (mixed with the stools, on the surface of the stool, or appears after passing the stool)? Melena results mostly from upper gastrointestinal bleeding; the stool is 'tarry' and shiny black with a characteristic odor. Distinguish this from the matt black stools associated with oral iron or bismuth therapy. Hemorrhoids and local anorectal diseases such as fissures will commonly present with passing small amounts of bright red blood per rectum. The blood is not mixed in the stools but is on the toilet paper, on top of the stools or in the toilet bowl.
4. What are the relieving factors e.g. drugs?
5. What are the associated symptoms e.g. hematemesis, weight loss, or jaundice?
6. What is the timing of the attack (course and duration)?
7. What are the exacerbating and the precipitating factors e.g. alcohol, corticosteroid or NSAID therapy?
8. What is the severity of the attack (amount and the frequency)?

CLINICAL CLUE

Melena signifies the loss of at least 60 ml of blood into the gastrointestinal tract. The amount of blood lost in hematochezia from upper gastrointestinal hemorrhage is usually large (more than a liter). Occult bleeding may reach 200 ml per day, cause iron deficiency anemia and signify serious gastrointestinal disease.

HOW CAN ESOPHAGEAL VARICES GRADE? (POSTGRADUATE STUDENTS)
The grades are based on variceal size and esophageal luminal narrowing
Grade I. Distended veins at the level of the mucosa.
Grade II. Isolated, straight varices that project into the lumen with no significant narrowing (< 5 mm).
Grade III. Large, tortuous varices that cause significant luminal narrowing (> 5 mm).
Grade IV. Almost complete luminal obstruction, signs of high bleeding risk.

CLINICAL CLUE

FOR POSTGRADUATE STUDENTS (CAUSES OF GI BLEEDING WITH NEGATIVE OGD)
The sources of gastrointestinal bleeding may not be readily identified via endoscopy for various reasons. Lesions may bleed intermittently, such as in the Dieulafoy lesion, a submucosal artery that protrudes through the mucosa and causes acute recurrent brisk hemorrhage. Volume contraction or a low hemoglobin concentration may alter the appearance of a bleeding source. For example, an esophageal varix may decompress with an episode of bleeding, or a site of angiodysplasia may not be evident in the setting of profound anemia. The source of bleeding may not originate in the intestinal lumen as is the case in hemosuccus pancreaticus and hemobilia. Finally, the cause of bleeding may be located within the small bowel and therefore not within the reach of the endoscope or colonoscope.

EXAMPLES

The condition started abruptly this morning as vomiting up of fresh blood. The patient had vomited three times; each comprising about one cupful of blood that was apparent in the first one and was associated with sweating, extreme weakness, and faintness. Of note the patient had a history of liver disease and one week history of passage of black tarry stools.

What is the diagnosis?
The diagnosis is upper gastrointestinal bleeding due to esophageal varices.

The condition started abruptly as vomiting up of fresh blood. The patient developed initially intense retching several times before he had vomited three times this morning but the blood was only apparent in the third vomitus and comprised about half cupful in amount. The condition was associated with sweating and faintness. Of note the patient had no prior history of liver disease or peptic diseases.

What is the diagnosis?
The diagnosis is upper gastrointestinal bleeding due to Mallory Weiss syndrome.

ABDOMINAL PAIN
There are four types of abdominal pain. These are visceral, parietal, referred, and psychogenic abdominal pain.
1. Visceral pain. Gut organs are insensitive to stimuli such as burning and cutting but are sensitive to distension, contraction, torsion and stretching. Visceral abdominal pain is a deep, poorly localized sensation. It is conducted via sympathetic splanchnic nerves. Pain from an unpaired structure, such as the pancreas, is felt in the midline and radiates through to the back. Pain from paired structures is felt on and radiates to the affected side, e.g. renal colic. Pain arising from foregut structures (stomach, pancreas, liver and biliary system) is localized above the umbilicus. Pain solely from the small intestine, e.g. small intestinal obstruction, is felt around the umbilicus (periumbilical). If the parietal peritoneum is involved, the pain will localize to that area, e.g. right iliac fossa pain in acute appendicitis and in Crohn's disease of the terminal ileum. Colonic pain can be felt either below the umbilicus, e.g. in the left iliac fossa from diverticular disease of the sigmoid colon, or in the upper abdomen, e.g. in the right hypochondrium from disease in the hepatic flexure. Both small and large bowels pain is colicky in nature; renal colic and biliary colic causes constant pain not colicky pain (misnamed).

2. Parietal pain. The parietal peritoneum is innervated by somatic nerves, and its involvement by disease processes, e.g. inflammation, infection or neoplasia, causes steady sharp, well-localised and lateralized parietal abdominal pain. The intensity of the pain is dependent on the type and amount of material to which the peritoneal surfaces are exposed in a given time period. For example, the sudden release into the peritoneal cavity of a small quantity of sterile acid like gastric juice causes much more pain than the same amount of grossly contaminated neutral feces. Enzymatically active pancreatic juice incites more pain and inflammation than does the same amount of sterile bile containing no potent enzymes. Blood and urine are often so bland as to go undetected if their contact with the peritoneum has not been sudden and massive. The pain of peritoneal inflammation is invariably accentuated by pressure or changes in tension of the peritoneum, whether produced by palpation or by coughing or sneezing. The patient with peritonitis lies quietly in bed, preferring to avoid motion, in contrast to the patient with colic, who may writhe incessantly.

3. Referred pain. Pain referred to the abdomen from the thorax, spine, or genitalia may prove a diagnostic problem because diseases of the upper part of the abdominal cavity such as acute cholecystitis or perforated ulcer are frequently associated with intrathoracic complications. An example of referred pain of thoracic origin is that diaphragmatic pleuritis from pneumonia or pulmonary infarction may cause pain in the right upper quadrant. In such a case, apparent abdominal muscle spasm caused by referred pain will diminish during the inspiratory phase of respiration, whereas it is persistent throughout both respiratory phases if it is of abdominal origin. Palpation over the area of referred pain in the abdomen also does not usually accentuate the pain and in many instances actually seems to relieve it. Referred pain from the spine, which usually involves compression or irritation of nerve roots, is characteristically intensified by certain motions such as cough, sneeze, or strain and is associated with hyperesthesia over the involved dermatomes. Pain referred to abdomen from testes or seminal vesicles is accentuated by slight pressure on either of these organs.

4. Psychogenic pain. In some patients, no organic cause can be found for abdominal pain despite investigation, and psychogenic causes (depression or somatisation disorder) may be responsible.

Some causes of abdominal pain are provided in BOX 1 – 26.

BOX 1 – 26. Some causes of abdominal pain

Surgical causes		Medical causes	
Appendicitis	Peptic ulcer	Abdominal aortic aneurysm	Lead toxicity
Diverticulitis	Diverticular disease	Ischemic bowel	Inflammatory bowel disease
Cholecystitis	Ovarian cyst	Gastroenteritis	Acute intermittent porphyria
Pelvic inflammatory disease	Aortic aneurysm	Hepatitis	Diabetic ketoacidosis
Pancreatitis	Intestinal obstruction	Familial mediterranean fever	Irritable bowel syndrome
Pyelonephritis	Biliary colic		
Intra-abdominal abscess	Uretric colic		

Non-alimentary causes of abdominal pain			
Myocardial infarction	Herpes zoster	Pleurisy	Salpingitis or tubal pregnancy
Dissecting aortic aneurysm	Cord compression	Diabetic ketoacidosis	Torsion of the testis/ovary

CLINICAL CLUE

Absence of tenderness and rigidity in the presence of continuous, diffuse pain in a patient likely to have vascular disease is quite characteristic of occlusion of superior mesenteric artery. Abdominal pain with radiation to the sacral region or genitalia should always signal the possible presence of a rupturing abdominal aortic aneurysm. This pain may persist over a period of several days before rupture and collapse occur.

Pain arising from the abdominal wall is usually constant and aching. Movement, prolonged standing, and pressure accentuate the discomfort and muscle spasm. Simultaneous involvement of muscles in other parts of the body usually serves to differentiate myositis of the abdominal wall from an intraabdominal process that might cause pain in the same region.

Whenever the cause of abdominal pain is obscure, a metabolic origin always must be considered. Pain of metabolic origin may simulate almost any other type of intraabdominal disease. In certain instances, such as hyperlipidemia, the metabolic disease itself may be accompanied by an intraabdominal process such as pancreatitis. C1 esterase deficiency associated with angioneurotic edema is often associated with episodes of severe abdominal pain. The pain of porphyria and of lead colic is usually difficult to distinguish from that of intestinal obstruction, because severe hyperperistalsis is a prominent feature of both. The pain of uremia or diabetes is nonspecific, and the pain and tenderness frequently shift in location and intensity.

PRESENTATION: ABDOMINAL PAIN

1. Where is the site of the pain? Visceral abdominal pain due to distension of hollow organs, excessive smooth muscle contraction, or mesenteric traction is poorly localised pain while parietal abdominal pain and abdominal wall pain is well localised.
2. What is onset of the abdominal pain e.g. sudden or gradual? In a previously asymptomatic patient, the sudden onset of severe abdominal pain, rapidly progressing to become generalized and constant, suggests perforation of a hollow viscus, a ruptured abdominal aortic aneurysm or mesenteric infarction. Preceding constipation suggests colorectal cancer or diverticular disease as the cause of perforation and prior dyspepsia suggests peptic ulceration. Co-existing peripheral vascular disease, hypertension, heart failure or atrial fibrillation may suggest a vascular disorder, e.g. aortic aneurysm or mesenteric ischemia. During the first hour or two after perforation, a 'silent interval' may occur when abdominal pain resolves transiently. The initial chemical peritonitis may subside before bacterial peritonitis becomes established.
3. What is the character (nature) of the pain? Constant pain means steady pain that usually arises from a solid organ, e.g. pancreatitis. Colicky pain means intermittent pain that lasts for a short period of time (seconds or minutes), eases off and then returns. It arises from hollow structures, e.g. small or large bowel obstruction. Biliary 'colic' are misnamed, as the pain is constant rather than colicky. Biliary colic is the term used for the pain associated with the temporary obstruction of the cystic or common bile duct by a stone usually migrating from the gall bladder. Biliary colic is misnamed as the pain rapidly increases to a peak intensity and persists over several hours (up to four hours) before gradually resolving. More protracted pain, particularly when lasting more than 4 hours and associated with fevers and rigors, suggests secondary complications such as cholecystitis, cholangitis or gallstone-related pancreatitis. Clinical differentiation (BOX 1 – 27) of biliary colic from acute cholecystitis is difficult but can usually be made from clinical and radiologic findings.

BOX 1 – 27. Clinical and radiological differentiations of biliary colic and acute cholecystitis

Clinical findings	Biliary colic	Acute cholecystitis
Right hypochondrial (upper quadrant) pain	Present	Present
Fever	Absent	Usually present
Duration of symptoms	<4 hr	> 6 hr
Abdominal tenderness	Absent or mild	Moderate to severe, especially over the liver and/or gallbladder (Murphy's sign)
Leukocytosis	Absent	Usually >11,000/μL
Ultrasound	Gallstones	Gallstones, thickening of the gallbladder wall

Other characters include stapping, dull-ache, constricting (squeezing), pricking, tearing, and heaviness in nature.

4. What is the radiation of the pain i.e. shifted or referred pain (BOX 1 – 28)? The term shifting pain means perception of pain in new site whilst the initial site of pain disappeared. The term referred pain means extension of pain to another site whilst the initial site of pain persisted.

BOX 1 – 28. Examples of pain radiation

The pain map	Radiation	Cause
From right or left iliac fossa to the corresponding testis	Referred	Urologic problem
From epigastrium to the back	Referred	Peptic ulcer
From right hypochondrium to the right shoulder	Referred	Gall bladder problem
From epigastrium where it disappeared then it appears in the right iliac fossa	Shifting	Appendicitis

In appendicitis, pain is initially localized around the umbilicus (visceral pain) and spreads as the inflammatory response progresses to involve the right iliac fossa (parietal or somatic pain). If the appendix ruptures, generalized peritonitis may develop. Occasionally, a localized appendix abscess develops, with a palpable mass and localized pain in the right iliac fossa.

5. What are the associated symptoms e.g. nausea or vomiting or headache?
6. What is the timing of the pain (duration, course, progression, and pattern)? Change in the pattern of symptoms suggests either that the initial diagnosis was wrong, or that complications have developed. In acute small bowel obstruction, a change from typical intestinal colic to persistent pain with abdominal tenderness suggests intestinal ischemia, e.g. strangulated hernia, an indication for urgent surgical intervention.
7. What are the precipitating, aggravating, and relieving factors? Precipitating factors refer to the factors that brought the pain where initially there was no pain; aggravating factors refer the factors that render the pain more severe where the pain was initially mild; and relieving factors refer to the factors that ease the pain or off it.

8. What is the severity of the pain? The severity of the pain is assessed by how much the pain interferes with sleeping, eating, or daily physical activity.

EXAMPLES

The condition started gradually over eight hours as an abdominal pain that was poorly localized in the area surrounding the umbilicus (i.e. Periumbilical) or some times in the area above it (i.e. epigastrium), mild in intensity, cramping in nature, lasted 6 hours, and associated with loss of appetite. The pain then SHIFTED to the right lower quadrant and became steady in character, more severe in intensity, aggravated by motion or cough, and associated with nausea, vomiting, and loss of appetite.

What is the diagnosis?
The diagnosis is acute appendicitis

The condition started gradually over few hours as an abdominal pain that was mild and tolerable discomfort at the start but soon became severe in intensity, constantly steady and boring in character, located in the area above the umbilicus and the area surrounding it, radiated to the back as well as to the chest, flanks, and lower abdomen, and associated with nausea, vomiting, and abdominal distention. The pain was more intense when the patient lied supine, and patients often obtained relief by sitting with the trunk flexed and knees drawn up. The patient gave a history of previous similar episodes all of which, like this attack, was precipitated alcoholic binge.

What is the diagnosis?
The diagnosis is acute pancreatitis

The condition started as an abdominal pain that occurred in the mid-evening time of the last night. The pain had followed fatty meal intake and was moderately severe in intensity but rapidly increased to peak intensity and persisted over several hours, constant in character, located in the right upper quadrant area, radiated to the right shoulder and right subscapular region, associated with nausea, vomiting, and fever, and slightly relieved with pain medication.

What is the diagnosis?
The diagnosis is acute cholecystitis

The condition started as an abdominal pain that occurred in the mid-evening time of the last night. The pain had followed fatty meal intake and was mild in intensity at the start but rapidly increased to peak intensity, persisted over two hours, and then spontaneously relieved. The pain was constant in character, located in the right upper quadrant area, radiated to the right shoulder and right subscapular region, associated with nausea and vomiting. The patient had previous similar attacks.

What is the diagnosis?
The diagnosis is biliary colic

The condition started gradually as an abdominal pain that was well localised to the left lower abdominal quadrant, intensely severe, cramping initially then steady in nature, lasted for hours, radiated to the flank and back, eased with medicine, and associated with loss of appetite, nausea, fever, and inability to pass stools or even gas.

What is the diagnosis?
The diagnosis is acute diverticulitis. Diverticulitis occurs when feces obstruct the neck of the diverticulum causing stagnation and allowing bacteria to multiply and produce inflammation and called left-sided appendicitis. This can then lead to bowel perforation, abscess formation, fistulae into adjacent organs, or even generalized peritonitis.

The condition started gradually as an abdominal pain that was severe in intensity, persisted over several hours, colicky in character, located in the mid-abdominal area, aggravated by food intake, and eased with medicine. The pain was associated with vomiting, abdominal distension, and inability to pass stools or gas. One note the vomiting initially contained bile and mucus then became feculent orange-brown in color with a foul odor. The patient had an old abdominal surgery.

What is the diagnosis?
The diagnosis is acute mechanical lower intestinal obstruction.
The mnemonic used for symptoms of intestinal obstruction collectively is PVCD where P refers to pain, V for vomiting, C for absolute constipation, and D for abdominal distension.

The condition started over the preceding two years as recurrent abdominal pain that was variable in intensity from mild enough to be ignored to severe interfering with daily activities apart from sleep, variable in location e.g. left abdomen, right abdomen, or lower part of the abdomen, frequently episodic and crampy bloating but mainly was constant in character, persisted over one to hours, exacerbated by eating, emotional stress, and premenstrual and menstrual, and improved by passage of flatus or stools. The pain was associated with bloating and abdominal distention.

What is the diagnosis?
The diagnosis is irritable bowel syndrome.

The condition started suddenly over the preceding minutes an abdominal pain that was diffuse all over the abdomen, severe interfering with daily activities, constant in character, persisted over one to two hours, worse with movement, and slightly improved by lying still. The pain was associated with nausea, vomiting, fever, and abdominal distention.

What is the diagnosis?
The diagnosis is ruptured viscus and peritonitis.

The condition started gradually over the preceding hours as an abdominal pain that was located in the left flank region, initially of moderate severity but 20 minutes later became so severe, colicky in nature, persisted over one to two hours, radiated to left groin and left testis, worse with movement that the patient cannot find a comfortable position, and slightly improved by pain medications. The pain was associated with nausea, vomiting, painful micturition, and passage of blood in urine.

What is the diagnosis?
The diagnosis is renal colic due to a stone that traverses the ureter. The vast majority of ureteral stones <0.5 cm in diameter will pass spontaneously.

The condition started suddenly over the preceding half an hour as an abdominal pain that was located in the left lower quadrant of the abdomen, so severe interfering with her daily activities, constant and some times stabbing in character, persisted over two hours, worse with any movement, and slightly improved by pain medications. The pain was associated with nausea, vomiting, and absence of menses for the previous two months and there was no radiation.

What is the diagnosis?
The diagnosis is ruptured ectopic pregnancy

CLINICAL CLUE

Intermittent abdominal pain precipitated by eating: think of obstruction (gastric outlet, small bowel); pancreatitis; ischemic bowel.

JAUNDICE

Jaundice refers to the yellowish discoloration of the skin, sclera and mucous membranes resulting from deposition of bilirubin in these tissues due to increased bilirubin concentration. Normal total bilirubin value is 0.12-1.0 mg/dl. Jaundice is usually detectable clinically when the plasma bilirubin exceeds 50 µmol/1 (3 mg/dl) but biochemically the patient is jaundiced when bilirubin exceeds 1mg/dl.

CLINICAL CLUE

1. When bilirubin level is less than 1 mg/dl the person had no jaundice.
2. When bilirubin level range from more than 1 mg/dl up to less than 3 mg/dl the patient had biochemical jaundice.
3. When bilirubin level is more than 3 mg/dl the patient had clinical jaundice.

The source of bilirubin is from hemoglobin breakdown (70 – 80%), catabolism of myoglobin and cytochromes enzymes and from ineffective erythropoiesis. Following erythrocytes destruction the hemoglobin (heme + globin) undergoes metabolic processes inside the body; globin is shifted into the protein pool of the body and heme is converted into iron and biliverdin. Biliverdin then is converted into bilirubin. This bilirubin is called unconjugated, indirect, and fat soluble (water – insoluble). Large amount of it binds to albumin to form bilirubin – albumin complex and the rest is called free bilirubin. Unconjugated bilirubin is not excreted in urine regardless of the height of its plasma concentration because its binding to albumin is too tight for effective glomerular filtration and it is not secreted by the tubules. The complex then reaches the liver where unconjugated bilirubin is conjugated by glucuronyl transferase to form conjugated, direct, and water soluble bilirubin that is then exported into the bile via specific carriers on the hepatocyte membrane. Although some bilirubin reaches the feces (stercobilinogen), most is converted to urobilinogen and to related compounds by bacteria within the ileum and colon, where the urobilinogen is reabsorbed, returns to the liver through the portal circulation, and is re-excreted into bile in a process of enterohepatic recirculation. Any urobilinogen that is not taken up by the liver reaches the systemic circulation loosely bound to albumin, from which it is cleared by the kidneys. Normal urine urobilinogen excretion is 4 mg/day or less.

CLINICAL CLUE

Ineffective erythropoiesis is defined as anemia with increased numbers of erythroid precursor cells in bone marrow but decreased numbers of mature circulating erythrocytes being released from bone marrow and therefore inappropriately low numbers of reticulocytes in blood. This condition is usually caused by defects in the maturation. Maturation disorders are divided into two categories: nuclear maturation defect that is associated with macrocytosis and cytoplasmic maturation defect that is associated with microcytosis. From practical point of view, the most common causes of anemia secondary to ineffective erythropoiesis are myelodysplasia, megaloblastic anemias, and thalassemias.

The differential diagnosis for yellowing of the skin is limited. In addition to jaundice, it includes carotenoderma, the use of the drug quinacrine, and excessive exposure to phenols. Carotenoderma (hypercarotemia) is the yellow color imparted to the skin by the presence of carotene; it occurs in healthy individuals who ingest excessive amounts of vegetables and fruits that contain carotene, such as carrots, leafy vegetables, squash, peaches, and oranges. Unlike jaundice, where the yellow coloration of the skin is uniformly distributed over the body, in carotenoderma the pigment is concentrated on the palms, soles, forehead, and nasolabial folds. Carotenoderma can be distinguished from jaundice by the sparing of the sclerae. Quinacrine causes a yellow discoloration of the skin in 4–37% of patients treated with it. Unlike carotene, quinacrine can cause discoloration of the sclerae.

Three types of jaundice are clinically identified. These are:

1. Prehepatic jaundice (BOX 1 – 29) due to hemolysis or some forms of congenital non-hemolytic hyperbilirubinemia. In hemolytic disorders the accompanying anemic pallor combined with jaundice may produce a pale lemon complexion. The stools and urine are normal in color. Gilbert's syndrome is the most common form of congenital non-hemolytic hyperbilirubinemia that causes unconjugated hyperbilirubinemia. In this syndrome serum liver enzyme concentrations are normal and jaundice is mild (plasma bilirubin <100 µmol/l) but increases during stress, fatigue, prolonged fasting, alcohol use, reduced caloric intake, and intercurrent illness. The diagnosis is made by identification of jaundice and normal liver function tests apart from slight elevation in serum unconjugated bilirubin. Provocation tests such as 48 h of fasting or the IV administration of nicotinic acid may be used.

BOX 1 – 29. Causes and clinical features of prehepatic jaundice

Causes of jaundice	Clinical features
Hemolysis	No stigmata of chronic liver disease other than jaundice Normal colored urine (if the cause is extravascular hemolysis) Normal colored stools Pallor due to anemia Splenomegaly due to increase reticuloendothelial activity
Gilberts syndrome (autosomal dominant)	No stigmata of chronic liver disease other than mild jaundice Normal colored urine Normal colored stools No pallor or splenomegaly

Hemolytic anemias are classified according to the site of destruction into intravascular and extravascular hemolytic anemia (BOX 1 – 30). In most hemolytic conditions red cell destruction is extravascular. The red cells are removed from the circulation by macrophages in the reticuloendothelial system, particularly the spleen (and therefore splenomegaly). When red cells are rapidly destroyed within the circulation (intravascular hemolysis), hemoglobin is liberated. This is initially bound to plasma haptoglobins but these soon become saturated. The hemoglobin is thus appearing in urine (hemoglobinuria) giving it a characteristic red dark color.

BOX 1 – 30. Causes of hemolytic anemias according to the site of destruction

Predominantly intravascular hemolysis		Predominantly extravascular hemolysis
Mismatched blood transfusion	Septicemia	Sickle cell and thalassemia
Microangiopathic hemolytic anemia	Cold autoimmune	Hereditary spherocytosis
March hemoglobinuria	hemolytic anemia	Hemolytic diseases of the newborn
Paroxysmal nocturnal hemoglobinuria (PNH)	Favism	Warm autoimmune hemolytic anemia

2. Hepatocellular jaundice (BOX 1 – 31) due to hepatic parenchymal diseases. Hepatocellular disease causes hyperbilirubinemia that is both unconjugated and conjugated. Urine will be dark and stools normal in color.

BOX 1 – 31. Causes and clinical features of hepatocellular jaundice

Causes of jaundice	Clinical features
Viral hepatitis Autoimmune hepatitis Hemochromatosis Wilson disease Alpha – 1 Antitrypsin deficiency Drugs and alcohol	Jaundice Dark colored urine Normal colored stools There may be stigmata of chronic liver diseases

3. Cholestatic or obstructive jaundice (BOX 1 – 32) due to intrahepatic or extrahepatic causes. In biliary obstruction, conjugated bilirubin in the bile does not reach the intestine, so the stools are pale. Conjugated bilirubin is water soluble and filtered by the kidney, so the urine is dark brown. Obstructive jaundice may be accompanied by generalized itch (pruritus) due to skin deposition of bile salts. Obstructive jaundice with abdominal pain is usually due to gallstones; if fever or rigors also occur (Charcot's triad), ascending cholangitis is likely. Painless obstructive jaundice suggests malignant biliary obstruction, e.g. cholangiocarcinoma or cancer of the head of the pancreas.

BOX 1 – 32. Causes and clinical features of cholestatic jaundice

Causes of jaundice		Clinical features	
INTRAHEPATIC CHOLESTASIS		Due to cholestasis	
Primary biliary cirrhosis	Pregnancy	Early features	Late features
Primary sclerosing cholangitis	Idiopathic recurrent cholestasis	Jaundice	Xanthelasma and xanthoma
Alcohol	Autoimmune hepatitis	Pale stools	Malabsorption that results in:
Drugs	Sever bacterial infections	Dark urine	• Weight loss
Viral hepatitis	Post – operative	Pruritus	• Steatorrhoea
Hodgkin's lymphoma	Cystic fibrosis		• Osteomalacia
			• Bleeding tendency
EXTRAHEPATIC CHOLESTASIS		Due to cholangitis (Charcot's triad)	
Carcinoma of ampulla, pancreas, and bile duct, and secondary metastatic depositions	Choledocholithiasis Parasitic infection Traumatic biliary strictures	Fever and rigors Right hypochondrial pain Jaundice	

CLINICAL CLUE

Charcot's triad is the combination of fever and rigors, right hypochondrial pain, and jaundice. Reynold's pentad includes shock and mental exhaustion in addition to Charcot's triad.

Remember that some forms of hepatocellular jaundice e.g. viral hepatitis, autoimmune hepatitis can result in cholestatic picture because of swelling of cells and edema resulting from the disease itself that may cause obstruction of the biliary canaliculi inside the liver (intra-hepatic cholestasis).

Plasma bilirubin, urine and stool analysis in patients with various types of jaundice is shown in BOX 1 – 33.

BOX 1 – 33. Plasma bilirubin, urine and stool analysis in jaundice

	Plasma bilirubin elevation	Urine			Stools
		Color	Bilirubin (bile pigment)	Urobilinogen	Color
Unconjugated	Indirect (unconjugated) fraction	Normal	-	++++	Normal
Hepatocellular	Direct and indirect fractions	Dark	++	++	Normal
Obstructive	Direct (conjugated) fraction	Dark	++++	-	Pale

ACUTE AND CHRONIC LIVER DISEASE

Acute liver failure is a clinical syndrome characterised by progressive deterioration in liver function and hepatic encephalopathy. It was previously further defined as that is occurring within 8 weeks of onset of the precipitating illness, in the absence of evidence of pre-existing liver disease. More recently, newer classification divides acute liver failure into hyperacute, acute and subacute according to the interval between onset of jaundice and encephalopathy. This classification is shown in BOX 1 – 34.

BOX 1 – 34. Classification of acute liver failure

Type	Time: jaundice to encephalopathy
Hyperacute	< 7 days
Acute	8-28 days
Subacute	29 days-12 weeks

When the interval between onset of jaundice and encephalopathy exceeds 12 weeks (previously 8 weeks) the term used is chronic liver failure. The term liver failure needs to be distinguished from liver disease (liver injury); liver disease is either acute (acute liver disease) or chronic (chronic liver disease). Acute liver disease may present with non-specific symptoms of fatigue and abnormal liver function tests (if liver damage is mild to moderate), or with jaundice and acute liver failure (if liver damage is severe). In chronic liver disease there are liver injury, inflammation and/or fibrosis occurring in the liver for more than 6 months (this duration is used to distinguish acute liver disease from chronic liver disease). Fibrosis is then progressed to cirrhosis. In the early stages of chronic liver disease patients can be asymptomatic (or with non-specific fatigue) with abnormal liver function tests (if liver damage is mild to moderate). With more severe liver damage, the presentation can be with features of cirrhosis (i.e. portal hypertension and liver insufficiency). Causes of acute liver disease and chronic liver disease are shown in BOX 1 – 35.

BOX 1 – 35. Causes of acute and chronic liver diseases

Acute liver disease		Chronic liver disease (and cirrhosis)	
Acute viral hepatitis (A, B, E)	Autoimmune hepatitis	Chronic viral hepatitis (B,C)	Wilson's disease
Drugs e.g. paracetamol	Wilson's disease	Alcoholic liver disease	α_1-antitrypsin deficiency
Toxins e.g. mushroom	Post shock or heart failure	Non-alcoholic fatty liver disease (NAFLD)	Primary biliary cirrhosis
Alcoholic liver disease	Liver metastasis	Hemochromatosis	Primary sclerosing cholangitis
Acute fatty liver in pregnancy	Cryptogenic		Cryptogenic

The difference between liver failure and liver disease is the presence of hepatic encephalopathy with biochemical evidences of deterioration in liver function; liver failure is thus represents the severe form of liver disease.

Causes of chronic liver disease can progress and lead to liver cirrhosis.

Patients with chronic liver disease have stigmata known as stigmata of chronic liver disease (BOX 1 – 36)

BOX 1 – 36. Stigmata of chronic liver disease

Signs of chronic liver disease	Signs of liver failure (hepatic encephalopathy)
Finger clubbing, leukonychia, palmar erythema	Flapping tremor of outstretched hands
Jaundice and anemia	Sweet smell on breath of fetor hepaticus
Men: gynaecomastia, testicular atrophy	Altered mental state
Women: breast atrophy	
Spider telangiectasia	
Ascites	
Splenomegaly and collateral vessels	
Hair loss, bruises, and purpura	

HOW TO PREDICT THE PRECENCE OF CHRONIC LIVER DISEASE AND CIRRHOSIS?

Although symptoms and signs are not particularly useful in identifying chronic hepatitis, biochemical and hematological blood test results are reliable. Serum bilirubin and albumin levels and the prothrombin time are normal in patients with chronic hepatitis, unless the disease is severe or advanced. Any elevation in serum direct bilirubin or decrease in albumin should be considered evidence of serious disease activity or injury. Serum aminotransferase levels (ALT > AST) can be normal when the disease is mild or inactive but can also be markedly elevated in the range typical of acute hepatitis (10 to 25 times the upper limit) during acute exacerbations. ALP and GGT are minimal in chronic hepatitis, unless cirrhosis is present. Creatine kinase and lactate dehydrogenase levels are normal. Serum immunoglobulin levels are mildly elevated or normal in chronic viral hepatitis but may be strikingly elevated in chronic autoimmune hepatitis. Blood counts are normal in chronic hepatitis, unless cirrhosis or portal hypertension is present with associated decreases in platelet counts and white blood cell counts. Serial determinations of platelet counts may provide the earliest clinical evidence of progression of chronic hepatitis to advanced fibrosis and cirrhosis. Blood tests results that suggest the presence of cirrhosis are low platelet counts (below 160,000), AST levels higher than ALT levels, elevations in serum bilirubin, decreases in serum albumin, prolongation of the prothrombin time, elevations in α-fetoprotein levels, and the presence of rheumatoid factor or high globulin levels.

Gut-derived neurotoxins that are not removed by the severely failing liver get to the brain and cause the symptoms that we know of as hepatic encephalopathy. Hepatic encephalopathy is a neuropsychiatric syndrome that is characterized by alterations in consciousness and behavior ranging from inversion of the sleep/wake pattern and forgetfulness (stage 1); to confusion, bizarre behavior, and disorientation (stage 2); to lethargy and profound disorientation (stage 3); to coma (stage 4). On physical examination, early stages may demonstrate only a distal tremor, but the hallmark of hepatic encephalopathy is the presence of asterixis (BOX 1 – 36). Precipitating factors for hepatic encephalopathy are shown in BOX 1 – 37.

BOX 1 – 37. Precipitating factors for hepatic encephalopathy (mnemonic: HEPATICS)

Hemorrhage in gastrointestinal tract/Hypokalemia	Trauma (including surgery)
Excess dietary protein	Infection
Paracentesis (or diuretics)	Constipation
Alkalosis/Anemia	Sedatives and antidepressants

PORTAL HYPERTENSION

Patients with chronic liver disease should be asked and checked for the clinical features or complications of portal hypertension and of cirrhosis. Liver cirrhosis will cause portal hypertension and liver insufficiency as shown below:

1 Portal hypertension
- 1 Hyperdynamic circulation
  - 1 Cirrhotic cardiomyopathy
  - 2 Hepatopulmonary syndrome
  - 3 Portopulmonary hypertension
  - 4 Congestive gastropathy and colopathy
- 2 Gastroesophageal varices
- 3 Ascites
  - 1 Hepatic hydrothorax

2 Hepatorenal syndrome
3 Spontaneous bacterial peritonitis
4 Hypersplenism
1 Low platelet
2 Low WBC
3 Low RBC
5 Hepatic encephalopathy
2 Liver insufficiency 1 Hepatic encephalopathy
2 Jaundice

The five primary complications of portal hypertension are hyperdynamic circulation, gastroesophageal varices, ascites, hypersplenism, and hepatic encephalopathy. Portal hypertension can occur as a part of liver cirrhosis or can occur alone without liver cirrhosis. Portal hypertension is defined as a portal venous pressure above 7 mmHg (normal portal venous pressure is 2 – 5 mmHg); the clinical features or complications of portal hypertension do not usually develop until the portal venous pressure exceeds 12 mmHg. The causes of portal hypertension are subcategorized into prehepatic, intrahepatic, and posthepatic causes as shown in FIGURE 1 – 5.

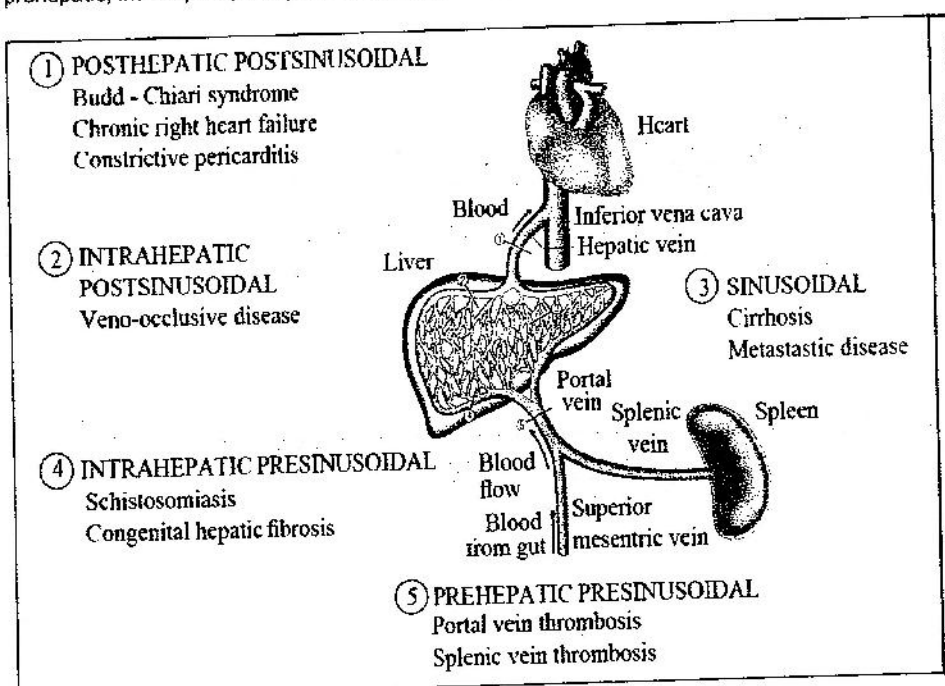


FIGURE 1 – 5. The classification and causes of portal venous hypertension.

Patients with prehepatic presinusoidal causes of portal hypertension have normal liver function. Therefore they complain of complications of portal hypertension without cirrhosis; almost all patients in the other categories may have features of cirrhosis. Cirrhosis is defined as a diffuse process characterized by fibrosis and the conversion of normal liver architecture into structurally abnormal nodules that lack normal lobular organization. This will interfere with liver blood flow and function and produces the clinical features of portal hypertension and impaired liver cell function.

CLINICAL CLUE

FOR POSTGRADUATE STUDENTS
In a patient with clinical evidence of portal hypertension (e.g., varices), the hepatic venous pressure gradient is useful in the differential diagnosis of the cause of portal hypertension: it will be normal in prehepatic causes of portal hypertension, such as portal vein thrombosis, and in intrahepatic but presinusoidal causes, such as schistosomiasis, but will be abnormal in sinusoidal causes of portal hypertension, such as cirrhosis, and in postsinusoidal causes, such as veno-occlusive disease.

CLINICAL
CLUE

FOR POSTGRADUATE STUDENTS

The hyperdynamic circulatory state in portal hypertension eventually results in high-output heart failure with decreased peripheral utilization of oxygen, a complication that has been referred to as cirrhotic cardiomyopathy. Vasodilation at the level of the pulmonary circulation leads to arterial hypoxemia, the hallmark of hepatopulmonary syndrome. Normal pulmonary capillaries are 8 μm in diameter, and red blood cells (slightly <8 μm) pass through them one cell at a time, thereby facilitating oxygenation. In hepatopulmonary syndrome, the pulmonary capillaries are dilated up to 500 μm, so passage of red cells through the pulmonary capillaries may be many cells thick. As a result, a large number of red cells are not oxygenated, which causes the equivalent of a right-to-left shunt. Conversely, portopulmonary hypertension occurs when the pulmonary bed is exposed to vasoconstrictive substances that may be produced in the splanchnic circulation and bypass metabolism by the liver. The diagnostic criteria for hepatopulmonary syndrome are arterial hypoxemia with a PaO_2 less than 80 mm Hg along with evidence of pulmonary vascular shunting on contrast echocardiography. The hepatopulmonary syndrome is characterized by platypnea and orthodeoxia, representing shortness of breath and oxygen desaturation that occur paradoxically upon assuming an upright position. Measurement of oxygen saturation by pulse oximetry is a reliable screening test for the presence of hepatopulmonary syndrome; the saturation of oxygen will be reduced by more than 5% on standing. Portopulmonary hypertension is diagnosed by the presence of mean pulmonary arterial pressure higher than 25 mm Hg on right heart catheterization, provided that pulmonary capillary wedge pressure is less than 15 mm Hg.

HOW TO APPROACH TO JAUNDICE?

S: Symtom O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Course and Duration) E: Exacerbating and precipitating factors S: Severity

SOCRATES

PRESENTATION: YELLOWISH DISCOLORATION OF SKIN, SCLERA, AND MUCOUS MEMBRANE

1. What are the symptoms that brought the patient e.g. jaundice (prehepatic, hepatic, or posthepatic), symptoms of liver failure (hepatic encephalopathy), or symptoms of mild to moderate chronic liver disease (non-specific fatigue), or symptoms of advanced chronic liver disease (symptoms of cirrhosis)?
2. What is the onset of jaundice e.g. acute or chronic?
3. What are the characters of the jaundice? Here ask the following four questions:
   - What is the color of skin, urine, and stools?
   - Is there any loss of appetite? Taste abnormalities (hypogeusia and dysgeusia) are recognized complications of liver disease. Smokers with acute hepatitis B and perhaps other acute liver diseases may lose interest in cigarettes because of a perverted sense of taste from liver inflammation. Impaired gustatory function with decreased sensitivity to bitter, salt, sweet, and sour taste has been reported in patients with cirrhosis.
   - Is there any weight loss (development of hepatoma)?
   - Does the patient have pruritus? Itching occurs with acute liver disease, appearing early in obstructive jaundice (from biliary obstruction) and somewhat later in hepatocellular disease (acute hepatitis). Itching also occurs in chronic liver diseases, typically the cholestatic forms such as primary biliary cirrhosis and sclerosing cholangitis where it is often the presenting symptom, occurring before the onset of jaundice. However, itching can occur in any liver disease, particularly once cirrhosis is present.
4. What are the relieving factors? Treatment of acute liver disease may simply include avoidance of the precipitating agents, provision of drugs in selected cases. In chronic liver disease this may include aspiration of abdominal ascites, shunt procedure, endoscopic banding for esophageal varices up to and reaching to transplantation.
5. What are the associated symptoms e.g. fever/rigor and abdominal pain (Charcot's triad) or abdominal swelling and hematemesis/melena (features of portal hypertension) or non-specific fatigue (symptoms of chronic liver disease) or other (BOX 1 – 38)? Fatigue is the most common and most characteristic symptom of liver disease. It is variously described as lethargy, weakness, listlessness, malaise, increased need for sleep. The fatigue of liver disease typically arises after activity or exercise and is rarely present or severe in the morning after adequate rest (afternoon vs. morning fatigue). Fatigue in liver disease is often intermittent and variable in severity from hour to hour and day to day.

BOX 1 – 38. The likely diagnosis in patients with jaundice "plus"

Clinical setting	The likely diagnosis
Jaundice and pneumonia	Mycoplasma pneumonae
Jaundice and pharyngitis	Viral hepatitis
Jaundice and parkinsonian features	Wilson's disease
Jaundice and hirsutism and acne	Autoimmune hepatitis
Jaundice and enlarged caudate lobe	Budd-Chiari syndrome
Jaundice, diabetes mellitus, and bronze skin	Hemochromatosis
Jaundice and diabetes mellitus	Hemochromatosis, nonalcoholic steatohepatitis, autoimmune hepatitis
Jaundice and emphysema	Alpha 1 Antitrypsin deficiency

6. What is the timing of the illness e.g. course and duration? Ask about any similar attacks
7. What are the exacerbating or precipitating factors? Here ask about history of operation e.g. blood transfusion, tattoo, tooth extraction (Hepatitis B or C), sexual contact (Hepatitis B or C), sick contact (Hepatitis A or E), family history of hemolytic anemia (congenital spherocytosis) or liver diseases (Wilson disease, hemochromatosis and α1 antitrypsin deficiency), history of hemolytic anemia (G6PD deficiency, sickle cell anemia, or congenital spherocytosis), drugs and alcohol, travel (Hepatitis A), and medical illnesses e.g. diabetes mellitus, obesity.
8. What is the severity of the attack?

Conjugated bilirubin, which is normally loosely bound to albumin, is readily filtered at the glomerulus; even modest degrees of conjugated hyperbilirubinemia result in bilirubinuria (positive bile pigment), which is always a pathologic finding. With prolonged conjugated hyperbilirubinemia, some of the conjugated bilirubin binds covalently to albumin and produces what is designated the delta bilirubin fraction. This type of bilirubin is not filterable by the glomerulus and does not appear in urine; it disappears slowly from plasma, with the 18- to 20-day half-life of the albumin (rather than the short half life of bilirubin of about 4 hours) to which it is bound. The prolonged half-life of albumin-bound conjugated bilirubin explains two previously unexplained enigmas in jaundiced patients with liver disease (1) that some patients with conjugated hyperbilirubinemia do not exhibit bilirubinuria during the recovery phase of their disease because the bilirubin is covalently bound to albumin and therefore not filtered by the renal glomeruli, and (2) that the elevated serum bilirubin level declines more slowly than expected in some patients who otherwise appear to be recovering satisfactorily.

WHAT ARE THE CAUSES OF SEVER RIGHT UPPER ABDOMINAL QUADRANT PAIN IN PATIENTS WITH JAUNDICE?
Severe right upper quadrant pain is most typical of gall bladder disease (cholangitis), liver abscess, and severe venoocclusive disease but is an occasional accompaniment of acute hepatitis.

WHAT ARE THE CAUSES OF DYSPNOEA IN PATIENTS WITH JAUNDICE?
Decreased exercise tolerance and shortness of breath in patients with liver disease may be caused by the hepatopulmonary syndrome, portopulmonary hypertension, and cardiomyopathy. Dyspnea can also result from impaired diaphragmatic excursion secondary to ascites.

CLINICAL CLUE

In hemolytic anemias e.g. sickle cell anemia, the total serum bilirubin is usually ≤ 6 mg/dL. Higher levels may occur when there is coexistent renal dysfunction, hepatocellular dysfunction (hepatitis B), in acute hemolysis such as a sickle cell crisis, or gall stone formation.

EXAMPLES

The condition started yesterday as a yellowish discoloration of the sclerae. The patient's family had repeatedly noticed that the patient's eye discolored yellow when fasting or emotionally upset. The patient had noticed repeated attacks and in each there was normal color of urine and stools. The patient had experienced no itching, no fever, no abdominal pain, and normal appetite. Prior to the attack there was no drug or alcohol intake, no sick or sexual contact, and no prior operation.

What is the diagnosis?
The diagnosis is Gilbert syndrome

The condition started yesterday as a yellowish discoloration of the sclerae. The patient's family had noticed that the patient's eye discolored yellow after two days of generalised weakness and abdominal pain. The patient had noticed repeated attacks of altered eye color when he ingested fava beans and in each there was dark color of urine but normal stools color. The patient had experienced no itching, no fever, and normal appetite. Prior to the attack there was no drug or alcohol intake, no sick or sexual contact, and no prior operation.

What is the diagnosis?
The diagnosis is G6PD deficiency. Dark colored urine owing to intravascular hemolysis.

The condition started as a yellowish discoloration of the skin and sclerae. The patient had noticed that he developed some sort of after noon weakness, nausea, headache, and muscle pain over the preceding two months. He developed one day after that yellowish discoloration of the skin and eyes and noticed dark color of urine but normal stools color. The patient had experienced mild itching especially at evening, low grade fever, and reduction in appetite. Prior to the attack there was no drug or alcohol intake, no sick contact, and no prior operation. Of note the patient had engaged in a non protected sexual contact six months ago.

What is the diagnosis?
The diagnosis is probably hepatitis B.

The condition started over the preceding one month as a yellowish discoloration of the skin and sclerae. He had noticed dark color of urine and pale stools. The patient had experienced generalised itching mostly at evening, no fever, and mild reduction in appetite. Prior to the attack there was no drug or alcohol intake, no sick or sexual contact, and no prior operation. The patient had noticed that he developed after noon fatigue, nausea, headache, and muscle pain over the preceding one month.

What is the diagnosis?
The diagnosis is cholestatic jaundice e.g. primary biliary cirrhosis.

ABDOMINAL DISTENTION (SWELLING)

Abdominal swelling may first be noticed by the patient because of a progressive increase in belt or clothing size, the appearance of abdominal or inguinal hernias, or the development of a localized swelling. Often, considerable abdominal enlargement has gone unnoticed for weeks or months, either because of coexistent obesity or because the abdominal distention has been insidious, without pain or localizing symptoms. Progressive abdominal distention may be associated with a sensation of "pulling" or "stretching" of the flanks or groins and vague low back pain. Tense abdominal swelling may produce increased intraabdominal pressure, resulting in indigestion and heartburn due to gastroesophageal reflux or dyspnea, abdominal wall hernias (inguinal and umbilical), orthopnea, and tachypnea from elevation of the diaphragm. The principal causes of abdominal distension are listed in BOX 1 – 39.

BOX 1 – 39. Causes of abdominal distension	
Factor	Consider
Fat	Obesity
Flatus	Pseudo-obstruction, obstruction
Feces	Subacute obstruction, constipation
Fluid	Ascites, tumours (especially ovarian), distended bladder
Fibroid	Tumours
Fetus	Check date of the last menstrual period
Functional	Bloating often associated with irritable bowel syndrome

HOW TO APPROACH TO ABDOMINAL DISTENSION?

S: Site O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Course and Duration) E: Exacerbating and precipitating factors S: Severity

SOCRATES

PRESENTATION: ABDOMINAL SWELLING

1. Where the site of distension is e.g. abdomen only or any other part is involved?
2. What is the mode of onset of abdominal distension e.g. sudden or gradual? Abdominal girth slowly increasing over months or years is usually due to obesity, but in a patient with weight loss it suggests intra-abdominal disease. More rapid abdominal distension may occur in patients with intestinal obstruction due to gaseous distention or in patients with Budd-Chiari syndrome.

CLINICAL CLUE

Hepatic vein thrombosis, which is the main cause of Budd-Chiari syndrome, may involve one, two, or all three of the major hepatic veins, with or without partial or complete occlusion of the inferior vena cava. Budd-Chiari syndrome can occur as a result of any process that interferes with the normal flow of blood out of the liver, including hypercoagulable state, constrictive pericarditis, malignancies (direct compression or invasion of hepatic veins, hypercoagulable state), infections (liver abscess), pregnancy, and membranous obstruction (webs) of the inferior vena cava are associated with Budd-Chiari syndrome. The syndrome may occur acutely or subacute and is characterized by the insidious onset of upper abdominal pain, hepatomegaly, and ascites.

3. What is the character of the abdominal distension e.g. constant or variable, painful or painless? Functional bloating is fluctuating abdominal distension that develops during the day and resolves overnight. It is particularly common in women and is rarely due to organic disease. It usually occurs in patients with irritable bowel syndrome. Chronic simple constipation rarely produces painful distension, unless associated with the irritable bowel syndrome. Painless abdominal distension in women may be the presenting symptom of ovarian pathology or a concealed pregnancy.
4. What are the relieving factors e.g. belching, vomiting or defecation? Recurrent abdominal distension that is characteristically relieved with defecation occurs in patients with irritable bowel syndrome.
5. What are the associated symptoms e.g. weight gain, fever, jaundice, or vomiting?
6. What is the timing of abdominal distension (duration and course)?
7. What are the exacerbating factors e.g. emotional stress or certain articles of food? Certain food articles such as legumes may produce gaseous abdominal distension.
8. What is the severity of the distension? The severity is assessed as to whether the swelling interferes with the patient's usual daily activities e.g. eating, walking, talking, and sleeping.

CLINICAL CLUE

DIAGNOSTIC CRITERIA FOR IRRITABLE BOWEL SYNDROME (ROME II CRITERIA)
Recurrent abdominal pain or discomfort at least 3 days per month in the last 3 months associated with two or more of the following:
1. Improvement with defecation.
2. Onset associated with a change in frequency of stool.
3. Onset associated with a change in form (appearance) of stool.

Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis.

EXAMPLE

The condition started gradually as progressive painless abdominal distension. The patient had noticed progressive increase in clothing size, pulling sensation in the flank, and mild low back pain over the preceding two weeks. This distension was not altered or relieved with belching or defecation. The patient had no similar attacks previously but he was a known case of liver disease. In the last days of his illness course he had developed a sense of breathlessness.

What is the diagnosis?
The diagnosis is ascites

The condition started over the preceding one week as progressive abdominal swelling without swelling in the other parts of the body. The patient had noticed progressive increase in clothing size, pulling sensation in the flank, and abdominal pain together with yellowish discoloration of the sclerae. This distension was not altered or relieved with belching or defecation. The patient had no similar attacks previously but she is currently on chemotherapy for blood disease.

What is the diagnosis?
The diagnosis is probably Budd-Chiari syndrome

HOW MUCH IS THE NORMAL AMOUNT PRESENT PHYSIOLOGICALLY IN PLEURAL, PERICARDIAL, AND PERITONEAL CAVITIES?

1. The pleural sac contains 5 – 10 cc of fluid.
2. The pericardial space contains about 15 – 50 cc of fluid.
3. The peritoneal cavity contains less than 100 cc of fluid.

DIARRHOEA

WHAT IS THE CHARACTER OF THE NORMAL STOOLS?
The normal frequency of population ranges from three bowel movements per day to one bowel action every third day and a normal stool consistency ranges from porridge – like to hard and pellety. The normal color of stool is brown color which is caused by stercobilin and urobilin, derivatives of bilirubin. The odor is caused principally by products of bacterial action; these products vary from one person to another, depending on each person's colonic bacterial flora and on the type of food eaten.

Gastroenterologists define diarrhea as the passage of more than 200 g of stool daily but clinically diarrhoea is the frequent passage of loose stools. Diarrhea persisting for more than 14 days is known as chronic diarrhea.
Two common conditions, usually associated with the passage of stool totaling <200 g/d, must be distinguished from diarrhea. These are pseudodiarrhea and fecal incontinence. Pseudodiarrhea, or the frequent passage of small volumes of stool accompanies irritable bowel syndrome or proctitis. Fecal incontinence is the involuntary discharge of rectal contents and is most often caused by neuromuscular disorders or structural anorectal problems. Overflow diarrhea may occur in nursing home patients due to fecal impaction that is readily detectable by rectal examination.
More than 90% of cases of acute diarrhea (BOX 1 – 40) are caused by infectious agents; these cases are often accompanied by vomiting, fever, and abdominal pain. The remaining 10% or so are caused by medications, toxic ingestions, ischemia, and other conditions. Most infectious diarrheas are acquired by fecal-oral transmission or, more commonly, via ingestion of food or water contaminated with pathogens from human or animal feces. Acute food poisoning is defined as the sudden onset of nausea, vomiting and diarrhoea after ingestion of a toxin. It is typically caused by Staphylococcal enterotoxin, Bacillus cereus or Clostridium perfringens. Traveler's diarrhoea may be defined as at least three loose stools to watery stools in 24 hour period with or without one or more of abdominal cramps, fever, nausea, vomiting, or blood in the stools. The so-called traveler's diarrhea is most commonly due to enterotoxigenic (ETEC) or enteroaggregative Escherichia coli (EAEC).

BOX 1 – 40. Causes of acute diarrhoea

Infectious		Non-infectious	
TOXIN-MEDIATED		GASTROINTESTINAL	
Bacillus cereus Staphylococcal enterotoxin	Clostridial spp.	Acute diverticulitis Inflammatory bowel disease	Bowel malignancy Overflow diarrhoea
INFECTIVE		METABOLIC UPSET	
Rotavirus gastroenteritis Norovirus Cholera	E. coli Infectious causes of bloody diarrhea (BOX 1 – 41)	Diabetic ketoacidosis Vasoactive intestinal peptide release	Carcinoid syndrome Uremia
PROTOZOAL		DRUGS AND TOXINS	
Giardiasis Amoebic dysentery Cryptosporidium	Isosporiasis	NSAIDs Cytotoxic agents Antibiotics	Plant toxins Heavy metals
SYSTEMIC ILLNESS			
Sepsis Meningococcal sepsis	Atypical pneumonia Malaria		

Causes of bloody diarrhoea are listed in BOX 1 – 41.

BOX 1 – 41. Causes of bloody diarrhoea

Infectious		Non-infectious	
Campylobacter spp. Clostridium difficile Enterohemorrhagic E. coli Entero-invasive E. coli Entero-toxogenic E. coli	Shigella dysentery Non-typhoidal salmonellae Vibro parahemolyticus Entamoeba histolytica (amoebic dysentery)	Anal fissure Bleeding hemorrhoids Colonic malignancy Diverticular disease	Inflammatory bowel disease Ischemic colitis Intussusception

ABCD/13

The incubation period and stereotypical histories (BOX 1 – 42) underlying acute diarrhea by infectious agents produces specific clinical features that may also be helpful in diagnosis as follows:

BOX 1 – 42. Some incubation period and stereotypical histories of infectious acute diarrhea

Incubation period	
1-6 hours	Staphylococcus aureus, Bacillus cereus
12-48 hours	Salmonella, E. coli
48-72 hours	Shigella, Campylobacter
> 7 days	Giardiasis, Amebiasis
Stereotypical histories	
Cholera	Profuse watery diarrhoea Severe dehydration resulting in weight loss
Giardiasis	Prolonged, non-bloody diarrhoea
Shigella	Bloody diarrhoea, vomiting, abdominal pain
Staphylococcus aureus	Severe vomiting with short incubation period
Escherichia coli	Commonest infectious agent amongst travelers Watery diarrhoea with abdominal cramps and nausea
Campylobacter	A flu-like prodrome is usually followed by crampy abdominal pain, fever and diarrhoea which may be bloody. Complication may include development of Guillain – Barré syndrome
Bacillus cereus	Two types of illness are seen: Vomiting within 6 hours, stereotypically due to rice Diarrhoeal illness occurring after 6 hours
Amebiasis	Gradual onset bloody diarrhoea, abdominal pain and tenderness that may last several weeks

Diarrhea persisting for more than two weeks is called chronic diarrhoea and warrants evaluation to exclude serious underlying pathology. In contrast to acute diarrhea, most causes of chronic diarrhea are noninfectious. The classification by pathophysiologic mechanism facilitates a rational approach to management, though many diseases cause diarrhea by more than one mechanism (BOX 1 – 43).

BOX 1 – 43. Causes of chronic diarrhoea

Secretory causes	Inflammatory causes
Stimulant laxatives (bisacodyl and senna) Chronic ethanol ingestion and other drugs and toxins Hormone-producing tumors (carcinoid syndrome, VIP-producing tumor, medullary thyroid cancer, mastocytosis, gastrinoma, colorectal villous adenoma) Addison's disease	Inflammatory bowel disease Lymphocytic and collagenous colitis Immune-related mucosal disease (1° or 2° immuno-deficiencies, food allergy, eosinophilic gastroenteritis) Infections (invasive bacteria, viruses, and parasites) Gastrointestinal malignancies
Osmotic causes	**Dysmotile causes**
Osmotic laxatives (lactulose and lactitol) Lactase and other disaccharide deficiencies	Irritable bowel syndrome Hyperthyroidism
Steatorrheal causes	**Iatrogenic causes**
Intraluminal maldigestion (pancreatic insufficiency, bacterial overgrowth, and liver disease) Mucosal malabsorption (celiac disease, infections, abetalipoproteinemia, ischemia) Post-mucosal obstruction (1° or 2° lymphatic obstruction)	Cholecystectomy Ileal resection Bariatric surgery Vagotomy Fundoplication surgery
Factitial causes	
Munchausen and eating disorders	

HOW TO APPROACH TO DIARRHOEA?

S: Subjective or objective O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Course and Duration) E: Exacerbating and precipitating factors S: Severity

SOCRATES

PRESENTATION: FREQUENT BOWEL MOTIONS OR FREQUENT LOOSE STOOLS

1. What does the patient mean by diarrhoea? Diarrhoea can be defined by patients in a number of different ways. Patients may complain of frequent stools (more than three per day being abnormal), of a change in consistency of the stools, which have become loose or watery, of fecal urgency (pseudodiarrhea), or of fecal incontinence.
2. What is the mode of onset of diarrhoea e.g. acute or chronic? Acute diarrhoea means diarrhoea lasting less than 14 days and chronic diarrhoea lasting than more 14 days?
3. What are the characters of the stools e.g. volume, shape (formed, unformed, or porridge –like), odor, and color (BOX 1 – 44)?

BOX 1 – 44. Causes of abnormal stool appearance

Stool appearance	Cause	Stool appearance	Cause
Fresh blood in or on stool	Large bowel, rectal or anal bleeding	Silvery	Steatorrhoea plus upper GI bleeding (pancreatic cancer)
Stool mixed with pus	Infective colitis or inflammatory bowel disease	Rice-water stool (watery with mucus and cell debris)	Cholera
Abnormally pale	Biliary obstruction	Black and tarry (melena)	Upper GI tract bleeding
Pale and greasy	Steatorrhoea	Grey/black	Oral iron or bismuth therapy

Secretory diarrhoea is likely if the diarrhoea is watery, painless, of high volume (commonly more than one liter per day) and persists when the patient fasts; there is no pus, mucus or blood, and the stools are not excessively fatty. Osmotic diarrhoea is characterised by its disappearance with fasting and by large volume stools related to the ingestion of food. Small bowel involvement is suggested when the bowel motion is of large volume, watery, not associated with blood but associated with abdominal bloating and cramping mid – abdominal pain while large bowel involvement is suggested when the bowel motion is of small volume and frequent and associated with blood and mucus in stool as well as cramping lower abdominal pain. Steatorrhoea is identified clinically when the stools are large volume, fatty or greasy, pale yellow to grey colored, extremely smelly, sometimes frothy and float in the toilet bowl (lavatory pan) and are difficult to flush away. Quantitatively, steatorrhea is defined as stool fat exceeding the normal 7 g/d; rapid-transit diarrhea may result in fecal fat up to 14 g/d; daily fecal fat averages 15–25 g with small intestinal diseases and is often >32 g with pancreatic exocrine insufficiency.

4. What are the relieving factors e.g. fasting, sleeping, or medications?

5. What are the associated symptoms e.g. local (mucus substance, pus, or blood) and systemic symptoms (abdominal cramps, tenesmus, fever, nausea and vomiting, or arthralgia? If there is blood with the stools does it mixed with or on the surface of the stool, or does it appear after passing the stool? Infectious diarrhoea is associated with the passage of pus with or without blood. Infectious diarrhea may be associated with systemic manifestations. Reiter's syndrome may accompany or follow infections by Salmonella, Shigella, Campylobacter, and Yersinia. Yersiniosis may also lead to an autoimmune-type thyroiditis, pericarditis, and glomerulonephritis. Enterohemorrhagic E. coli (O157:H7), Shigella dysenteriae type 1, Vibrio parahemolyticus, and Clostridium difficile can lead to the hemolytic-uremic syndrome with an attendant high mortality rate.
6. What is the timing of the diarrhoea (day or night time, course, and duration)? Diarrhoea occurring at night suggests organic disease.
7. What are the precipitating and/or exacerbating factors? Diarrhea closely following food consumption at a picnic, banquet, or restaurant may suggest infection with Salmonella, Campylobacter, or Shigella from chicken; enterohemorrhagic E. coli (O157:H7) from undercooked hamburger; Bacillus cereus from rice; Staphylococcus aureus or Salmonella from mayonnaise or creams; and Salmonella from eggs.
8. What is the frequency (number of motion/day)? Passage of more than three loose bowel motions is abnormal.

EXAMPLES

The condition started acutely over the previous two day as frequent loose stools. The bowel motion was watery at the start and associated with abdominal discomfort and low grade fever. The motion then occurred 8 times per day, in both day and night time, was yellow to green in color, was of small volume, and associated with passage of mucus and blood with it. His condition had been associated with fever, abdominal cramps, and nausea and vomiting.

What is the diagnosis?
The diagnosis is infective bloody diarrhoea e.g. shigellosis.

The condition started gradually over the previous one week as frequent loose stools. The bowel motions occurred initially 4 times per day and were associated with abdominal discomfort and low grade fever. The motion then occurred 10 – 12 times per day, in both day and night time, was yellow to green in color, was of small volume and unformed, and associated with passage of mucus, pus and blood with it. His condition had been associated with fever, abdominal cramps and feeling of incomplete defecation, and nausea and vomiting.

What is the diagnosis?
The diagnosis is infective bloody diarrhoea e.g. amebiasis.

The condition started intermittently over the previous three months as frequent loose stools. Each time the attack occurred over three days that was followed by a period of normal motion and then attack recurred again. The bowel motion occurred 5 times per day, in both night and postprandial time, green in color, of small volume, liquid unformed, and associated with passage of mucus, pus and blood that was mixed with the stools. His condition had been associated with fever, abdominal cramps and feeling of incomplete defecation. This attack was the 9th over these three months.

What is the diagnosis?
The diagnosis is chronic bloody diarrhoea e.g. inflammatory bowel disease (ulcerative colitis).

The condition started suddenly over the previous six hours as frequent loose stools. The attack involved the patient and other six patients from the same region. The bowel motion occurred very frequent about 20 times over the previous six hours, rice-watery, voluminous (large volume), painless and non offensive, liquid unformed, and was slightly cloudy with flecks of mucus but no blood. His condition had been associated with muscle weakness and vomiting that followed the attack. There was no fever or abdominal cramps.

What is the diagnosis?
The diagnosis is acute watery diarrhoea; most likely cholera.

The condition started suddenly, four hours after food intake at restaurant as vomiting and frequent loose stools. The attack involved the patient and his three friends who all ate processed meat and salad from the same restaurant. The bowel motion was mild and occurred two times, was watery, small volume, liquid unformed, and was not associated with mucus or blood. His condition had been associated with mild abdominal pain and severe vomiting.

What is the diagnosis?
The diagnosis is probably staphylococcal food poisoning.

CONSTIPATION

Constipation refers to infrequent defecation (less than two bowel movements weekly), straining to have a bowel movement, and passage of hard stools that are difficult to evacuate. Patients may also complain of sensation of incomplete evacuation and either perianal or abdominal discomfort. Causes of constipation are shown in BOX 1 – 45.

BOX 1 – 45. Causes of constipation

Gastrointestinal disorders		Non-gastrointestinal disorders	
DIETARY		DRUGS	
Lack of fiber and /or fluid intake		Opiates Anticholinergics Calcium antagonists	Iron supplements Aluminum – containing antacids
MOTILITY		NEUROLOGICAL	
Irritable bowel syndrome	Chronic intestinal pseudobstruction	Multiple sclerosis Cerebrovascular accidents	Spinal cord lesions Parkinsonism
STRUCTURAL		METABOLIC /ENDOCRINE	
Colonic carcinoma Hirschsprung`s disease	Diverticular disease	Diabetes mellitus Hypothyroidism	Hypercalcemia Pregnancy
DEFECATION		OTHER	
Anorectal disease (Crohn's, fissures, hemorrhoids)		Immobility	Depression

HOW TO APPROACH TO CONSTIPATION?

S: Subjective or objective O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Course and Duration) E: Exacerbating and precipitating factors S: Severity

SOCRATES

PRESENTATION: INABILITY TO PASS STOOLS OR PASSAGE OF HARD STOOLS

1. What does the patient mean by constipation? Constipation can refer to the passage of infrequent stools, hard stools or stools that are difficult to evacuate. Difficulty with evacuation of feces may occur with disorders of the pelvic floor muscles or nerves, or anorectal disease (e.g. fissure, or stricture). Patients with this problem may complain of straining, a feeling of anal blockage or even the need to self-digitate.
2. What is the mode of onset of constipation e.g. sudden or gradual? Constipation can arise after partial colonic obstruction from carcinoma; it is, therefore, very important to determine whether there has been a recent change in bowel habit, as this may indicate development of a malignancy.
3. What are the characters of the stool e.g. shape and color? Hard pellety stools occur with slow transit while loose watery stools are associated with rapid transit. Both small pellety or very large stools are more difficult to expel than normal stools.
4. What are the associated symptoms e.g. straining, abdominal cramps, blood, or diarrhoea? A chronic but erratic disturbance in defecation (typically alternating constipation and diarrhoea) associated with abdominal pain, in the absence of any structural or biochemical abnormality, is very common; such patients are classified as having the irritable bowel syndrome.
5. What is the timing of constipation (course and duration)?
6. What are the exacerbating and precipitating factors e.g. drugs, food, or stress?
7. What is the severity of the attack? Severity is assessed as whether the attack interferes with the patient's usual daily activities such as sleeping or eating.

CLINICAL CLUE

Anismus/dyschezia means difficulty emptying the rectum despite prolonged straining while tenesmus means persistent urge to empty the rectum with the feeling of incomplete evacuation.

EXAMPLE

The condition chronically started as months of infrequent passage of stools alternating with only brief period of frequent loose stools. The patient had noticed passage of hard pellety narrowed-caliber stools that were associated with passage of mucus but not blood or pus and were of normal color. The condition was exacerbated during stressful event and associated with belching, flatulence, and mild abdominal pain that were relieved with defecation.

What is the diagnosis?
The diagnosis is constipation – predominant irritable bowel syndrome.

HEMATURIA

Hematuria may be visible and reported by the patient (macroscopic hematuria) or invisible and detected on dipstick testing of urine (microscopic hematuria). Microscopic hematuria is defined as two to five RBCs per high-power field and can be detected by dipstick. Presence of blood in the urine indicates bleeding from any where in the urinary tract. Distinguish hematuria from contamination of the urine by blood from the female genital tract during menstruation. Hematuria should be differentiated from other causes of red colored urine (BOX 1 – 46).

BOX 1 – 46. Causes of red colored urine

Hematuria	Bilirubinuria e.g. obstructive jaundice
Hemoglobinuria	Food dyes e.g. beetroot
Myoglobinuria	Drugs (phenolphthalein, senna, rifampicin, and levodopa)
Alkaptonuria	Porphyria

PRESENTATION: PASSAGE OF BLOOD IN URINE

1. Does it menstrual blood or true hematuria?
2. What is the amount of blood that passes with the urine?
3. Is it painful or painless (BOX 1 – 47)?

BOX 1 – 47. Causes of hematuria

Painless	Painful	May be either
Glomerulonephritis	Urinary tract infection	Urinary tract infection
Tumours of the kidney, ureter, bladder or prostate	Renal stones with obstruction	Reflux nephropathy and renal scarring
Tuberculosis or schistosomiasis	Loin pain-hematuria syndrome	Adult polycystic kidney disease
Hypertensive nephrosclerosis		Renal stones without obstruction
Interstitial nephritis (unless very acute/severe)		
Acute tubular necrosis		
Renal ischemia (renovascular disease)		
Distance running or severe exercise		
Coagulation disorders, anticoagulant therapy		

4. Is it initial, terminal or total? Schistosomiasis can cause terminal hematuria.
5. Has the patient had taken anticoagulant drugs e.g. warfarin or heparin or drugs that discolor the urine?
6. Has the patient had any systemic illness e.g. bleeding tendency, renal stone former, or hypertension?
7. Had the patient had exposed to trauma?
8. What are the associated features e.g. fever, weight loss, night sweat?

Other abnormalities of urine should be noted as color changes, amount, frequency, and association with pain or other systemic symptoms. Possible causes of color changes in the urine are provided in BOX 1 – 48.

BOX 1 – 48. Causes of altered urine color

Milky	Smoky pink	Faomy	Blue or green
Acid urine: urate crystals Alkaline urine: insoluble phosphate Infection: pus Chyluria	Hematuria (>0.54 ml blood/l urine)	Proteinuria	Pseudomonas urinary tract infection Bilirubin Methylene blue

The dipstick test is positive on exposure to hemoglobin, myoglobin, or intact RBCs. The presence of myoglobin or hemoglobin is suspected if the dipstick is intensely positive and there are few RBCs in the sediment on microscopic examination.

EXAMPLE

The condition started three weeks ago when the patient had noticed passage of blood with urine. Urination was not painful and totally discolored red. The condition was associated with weight loss, intermittent low-grade fever, and abdominal pain. The patient had no prior such an attack, no medical illness, and on no any medication.

What is the diagnosis?
The diagnosis is painless hematuria e.g. renal cell carcinoma.

JOINT PROBLEMS

This section is specially designated for fifth medical students and elder students. The principal clinical complaints of joint disease are joint pain, stiffness, swelling, or limitation of joint movement. Causes of arthritis are wide and include:

1. Infection. In adults the most likely organism is Staphylococcus aureus, particularly in patients with rheumatoid arthritis and diabetes. In young, sexually active adults disseminated gonococcal infection is an important cause. Amongst the elderly or those who misuse intravenous drugs, Gram-negative bacilli or other microorganisms are important.
2. Seropositive arthritis e.g. rheumatoid arthritis.
3. Seronegative arthritis (BOX 1 – 49).

BOX 1 – 49. Causes of seronegative spondarthritides

Ankylosing spondylitis
Reactive arthritis (Reiter's syndrome)
Psoriatic arthropathy
Arthritis associated with inflammatory bowel disease (Crohn's disease, ulcerative colitis)

4. Connective tissue diseases e.g. systemic lupus erythematosus, systemic sclerosis, and Sjögren's syndrome.
5. Crystal-associated diseases e.g. gout and pseudogout.
6. Malignancy e.g. leukemia, lymphoma, and hypertrophic osteoarthropathy.
7. Vasculitis e.g. polyarteritis nodosa and cryoglobulinemia.
8. Drugs e.g. corticosteroid withdrawal, glibenclamide, methyldopa, ciclosporin, isoniazid, and barbiturates.
9. Systemic diseases (BOX 1 – 50).

BOX 1 – 50. Systemic diseases that cause arthritis

Gastrointestinal diseases	Hemochromatosis, autoimmune hepatitis, primary biliary cirrhosis, gluten-sensitive enteropathy, and inflammatory bowel disease
Hematological disorders	Hemophilia, hemoglobinopathies, and plasma cell dyscrasias
Endocrine disorders	Diabetes mellitus, thyroid disorders, parathyroid disorders, and acromegaly
Other	Sarcoidosis, relapsing polychondritis, and cystic fibrosis

PRESENTATION: LIMITATION OF JOINT MOVEMENT OR JOINT PAIN

HOW TO APPROACH TO JOINT PROBLEM?

SOCRATES

S: Site O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Course and Duration) E: Exacerbating and precipitating factors S: Severity

1. Where is the site of the complain e.g. articular or non-articular (FIGURE 1 – 6)? Distinguishing between articular and nonarticular conditions requires a careful and detailed examination. Articular structures include the synovium, synovial fluid, articular cartilage, intraarticular ligaments, joint capsule, and juxtaarticular bone. Nonarticular (or periarticular) structures, such as supportive extraarticular ligaments, tendons, bursae, muscle, fascia, bone, nerve, and overlying skin, may be involved in the pathologic process. Articular disorders may be characterized by deep or diffuse pain, pain or limited range of motion on active and passive movement, and swelling (caused by synovial proliferation, effusion, or bony enlargement), crepitation, instability, "locking," or deformity. By contrast, nonarticular disorders tend to be painful on active, but not passive (or assisted) motion, demonstrate point or focal tenderness in regions adjacent to articular structures, and have physical findings remote from joint capsule. Moreover, nonarticular disorders seldom demonstrate swelling, crepitus, instability, or deformity.

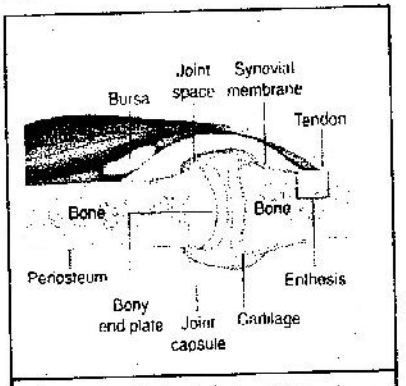


Figure 1 – 6. Anatomical joint structure.

2. What is the mode of onset duration of the complain e.g. acute versus chronic? The duration of acute arthritis is less than 6 weeks while that of chronic arthritis is more than 6 weeks.
3. What are the characters of the attack? Determine the following:
   - Is it inflammatory or non-inflammatory? With inflammatory joint complain the patients will usually have prolonged morning stiffness (more than 1 hour), local signs of inflammation (hotness, redness, tenderness, swelling, and loss of function), and systemic symptoms in the form of fever and weight loss. Intermittent stiffness is associated with non-inflammatory conditions (e.g. osteoarthrosis) and usually lasts <60 min.

Pain belonging to the musculoskeletal system is divided into:

1. Usage pain. This pain is worse on use and relieved by rest. It indicates a mechanical strain.
2. Rest pain. This pain is worse after rest and improved by movement. It indicates an inflammation.
3. Night or 'bone' pain. This pain mostly occurs at night and poorly related to movement. It indicates a bone origin.

- How many joints (BOX 1 – 51) involved in the attack e.g. monoarticular (one joint), oligoarticular or pauciarticular (2 – 4 joints) or polyarticular (more than 4 joints)?

BOX 1 – 51. Causes of arthritis according to the number of joints involved

Monoarthritis	Oligoarthritis	Polyarthritis
Septic arthritis	Osteoarthrosis (most common)	Rheumatoid arthritis
Gout and pseudogout	Seronegative spondarthritis	Lupus
Trauma	Infection as in bacterial endocarditis	Systemic sclerosis
Hemarthrosis in clotting abnormality	Juvenile idiopathic arthritis	Polymyositis
Foreign body (e.g. plant thorn)	Oligoarticular presentation of polyarthritis	Viral arthritis
Monoarticular presentation of polyarthritis		Seronegative spondarthritis
		Chronic gout
		Hemochromatosis
		Acromegaly

- When multiple joints are involved what is nature of involvement e.g. migratory or additive. Migratory arthritis means involvement of new joint and the previous one had been improved (BOX 1 – 52). Additive means involvement of new joint and the previous one still involved.

BOX 1 – 53. Causes of migratory arthritis		
Gonococcal arthritis	Relapsing polychondritis	Crohn's disease
Mumps arthritis	Leukemia	Whipple's disease
Acute rheumatic fever	Hyperlipidaemia	

- Does the arthritis involve the axial or peripheral joints? If peripheral determine symmetry, small/large joints, predominance of upper/lower limbs (BOX 1 – 54).

BOX 1 – 54. Characteristic features of some polyarthritis	
Cause	Characteristics
Rheumatoid arthritis	Symmetrical, small and large joints, upper and lower limbs
Seronegative arthritis	Asymmetrical, large > small joints, lower > upper limbs, spondylitis
Lupus	Symmetrical, small > large joints, joint damage uncommon
Systemic sclerosis and polymyositis	Symmetrical, small and large joints
Juvenile idiopathic arthritis	Symmetrical, small and large joints, upper and lower limbs
Chronic gout	Distal > proximal joints, preceded by acute attacks
Viral arthritis	Very acute, self-limiting

4. What are the relieving factors e.g. rest, exercise, work?
5. What are the associated features (extra-articular manifestations)? (see BOX 1 – 55)

BOX 1 – 55. Some extra-articular features of arthritis	
Clinical feature	Disease association
Psoriasis, nail pitting and dystrophy	Psoriatic arthritis
Raynaud's phenomenon	Systemic sclerosis, lupus, dermatomyositis, polymyositis, Sjögren's
Splinter hemorrhages, nail-fold infarcts	Vasculitis, endocarditis
Oral ulcers	Lupus, reactive arthritis, Behçet's syndrome
Large nodules (mainly extensor surfaces)	Rheumatoid arthritis, rheumatic fever, chronic tophaceous gout
Clubbing	Enteropathic arthritis, metastatic lung cancer, endocarditis
Uveitis	Seronegative spondarthritis
Episcleritis, scleritis	Rheumatoid arthritis, vasculitis
Pleuro-pericarditis	Lupus, rheumatoid arthritis
Fibrosing alveolitis	Rheumatoid arthritis, lupus, other connective tissue disease
Hepatosplenomegaly	Rheumatoid arthritis, lupus
Hematuria, proteinuria	Lupus, vasculitis, systemic sclerosis
Fever, lymphadenopathy	Infection, lymphoma, systemic juvenile idiopathic arthritis

6. What is the timing of the attack (course and duration)?
7. What are the exacerbating and precipitating factors e.g. activity, rest, operation, drugs, sexual contacts, or dysentery? Reiter's disease or reactive arthritis typically develops 1-3 weeks following sexual exposure or an attack of dysentery. Gouty attack may follow operation.
8. What is the severity of the attack? Severity is determined as to whether pain interferes with daily activities like sleeping, eating, etc?

EXAMPLES

The condition started gradually over the preceding year as mild joint pain and limitation of movement. The patient had noticed that both knee joints had been affected simultaneously and symmetrically but no redness or hotness, and with ten minutes of early morning stiffness. Her joints became better with rest and worse with movement. The patient had not experienced fever or weight loss and there was no prior history of sexual contact, bloody bowel motion, drugs or alcohol intake.

What is the diagnosis?
The diagnosis is osteoarthritis of the knee joints.

The condition started gradually over the preceding three days as joint pain and limitation of movement. The patient had noticed that the right knee joint had become red, hot, swollen, and painful, with severe limitation in its movement. Her joint became better with rest and worse with any joint movement. The patient had experienced high grade fever with loss of appetite. Prior to the attack, there was no prior history of joint problem, sexual contact, bloody bowel motion, drugs or alcohol intake.

What is the diagnosis?
The diagnosis is septic arthritis of the right knee joint.

The condition started gradually over the preceding eight weeks as joint pain and limitation of movement. The patient had noticed that her right and left wrist joints and finger joints had become, hot, swollen, and painful, with severe limitation in its movement that incapacitated her to perform her daily activities in the early morning. Her condition then deteriorated when her ankle joints had also been involved in an additive manner. She experienced one and half hour of early morning stiffness and noticed that her joint became better after work and with the use of pain medications and worse after rest. The patient had experienced intermittent low-grade fever, red painful eyes with reduction of appetite. Prior to the attack, there was no prior history of joint problem, sexual contact, bloody bowel motion, drugs or alcohol intake.

What is the diagnosis?
The diagnosis is rheumatoid arthritis.

The condition started gradually over the preceding three months as joint pain and limitation of movement. The patient had noticed that his right hip and right knee joints had become hot, swollen, and painful, with severe limitation in its movement that incapacitated her to perform her daily activities in the early morning. Her condition then deteriorated when her left ankle joint and right elbow had also been involved in an additive manner. She experienced one and half hour of early morning stiffness and noticed that her joint became better after work and with the use of pain medications and worse with rest. The patient had experienced intermittent low-grade fever with mild reduction of appetite. Prior to the attack, there was no prior history of joint problem, sexual contact, bloody bowel motion, drugs or alcohol intake. Of note the patient had chronic history of skin disease.

What is the diagnosis?
The diagnosis is seronegative psoriatic arthropathy.

WHAT ARE THE CAUSES OF MORNING STIFFNESS?

1. Inflammatory arthritis (> 1 hour)
2. Degenerative arthritis (< 1 hour)
3. Myxedema
4. Polymyalgia rheumatica

CLINICAL CLUE

The classic triad of Reiter's disease is non-specific urethritis, conjunctivitis, and reactive arthritis. Reactive arthritis is precipitated by (mnemonic CCYSS) bacterial dysentery-mainly Salmonella, Shigella, Campylobacteror Yersinia, or sexually acquired infection with Chlamydia

MUSCLE WEAKNESS

Muscle weakness can arise from (BOX 1 – 56) a variety of metabolic, endocrine, infective and neurological causes.

BOX 1 – 56. Causes of muscle weakness

Endocrine		Metabolic
Hypothyroidism Hyperthyroidism Osteomalacia	Cushing's syndrome Addison's disease Diabetes mellitus	Hypokalaemia Glycogen storage diseases

Drugs/toxins		Infections
Alcohol	Amiodarone	Viral (HIV, CMV, rubella, Epstein-Barr)
Fibrates	Statins	Bacterial (Clostridia, staphylococci, tuberculosis, Mycoplasma)
Steroids	ß-blockers	Parasitic (schistosomiasis, toxoplasmosis)

Neurological		
1	Supraspinal lesions	Stroke and other structural lesions
2	Spinal cord lesions	See Box 7 – 19
3	Anterior horn cell disorders	Poliovirus
4	Peripheral neuropathy (motor)	See Box 7 – 21
5	Neuromuscular junction disorders	Myasthenia gravis, Lambert Eaton syndrome, and botulism

Muscle disorders		
1	Inflammatory	Polymyositis, dermatomyositis, and inclusion body myositis
2	Muscle dystrophy	See Box 7 – 12

PRESENTATION: MUSCLE WEAKNESS OR MUSCLE PAIN

HOW TO APPROACH TO MUSCLE WEAKNESS?

S: Site O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Course and Duration) E: Exacerbating and precipitating factors S: Severity

SOCRATES

1. Where is the site of weakness e.g. proximal or distal muscle weakness? Proximal muscle weakness usually indicates a muscle disease (myopathy) which typically causes difficulty with standing from a seated position, squatting and lifting overhead. Distal power, such as hand grip, is usually preserved while distal muscle weakness usually indicates a neurological cause (neuropathy) which is even more likely if there are sensory abnormalities or if the weakness is unilateral or focal.
2. What is the mode of onset e.g. acute, subacute, or chronic? An abrupt onset suggests a vascular disturbance, such as a stroke, or certain toxic or metabolic disturbances, whereas subacute onset of days to weeks is commonly associated with a neoplastic, infective, or inflammatory process. Weakness that evolves slowly over several months or years often has a hereditary, degenerative, endocrinologic, or neoplastic basis.
3. What is the character of the weakness?
   - Is it subjective feeling of fatigue or objective 'true' weakness with loss of muscle power and function? The former is a non-specific manifestation of many organic diseases and also of psychological distress.
   - Is it muscle pain with weakness or muscle pain with stiffness? The former is associated with intrinsic muscle diseases while the latter is typical of polymyalgia rheumatica and may be confused with the symptoms of large joint disease.
   - Is it intermittent or persistent? Disorders causing intermittent weakness include myasthenia gravis, periodic paralyses (hypokalemic, hyperkalemic, and paramyotonia congenita), and metabolic energy deficiencies while persistent muscle weakness include most types of muscular dystrophy, polymyositis, and dermatomyositis.
   - Is it ascending or descending? The former suggests Guillain Barré syndrome while the latter suggests botulism.

CLINICAL CLUE

The five "Ds" describe the major symptoms of botulism: (1) diplopia; (2) dysphonia; (3) dysarthria; (4) dysphagia; and (5) descending, symmetric flaccid paralysis that develops 12 to 72 hours after exposure.

4. What are the relieving factors? The weakness of myasthenia gravis is characteristically and typically improved with rest while that of Lambert Eaton syndrome is improved with exercise.
5. What are the associated symptoms? Associated sensory loss may suggest neuropathic cause of weakness. Endocrine and metabolic causes have many associated symptoms.
6. What is the timing of the weakness (course and duration)? Myasthenia gravis characteristically has diurnal variation with the patients being well in the morning and worsen at the end of the day.
7. What are the exacerbating and the precipitating factors? The weakness of myasthenia gravis is characteristically worsened by repeated exertion (fatigability) and improved by rest; exercise and cold may precipitate weakness in paramyotonia congenita (BOX 1 – 57); carbohydrate meal ingestion may precipitate hypokalemic periodic paralysis; potassium ingestion may precipitate hyperkalemic periodic paralysis.

BOX 1 – 57 Some groups of channelopathies

Channel	Muscle disease	Clinical features
Sodium	Paramyotonia congenita	Cold-evoked myotonia with episodic weakness provoked by exercise and cold
	Hyperkalemic periodic paralysis	Brief, frequent episodes of weakness triggered by potassium ingestion
	Hypokalemic periodic paralysis	Episodic weakness triggered by carbohydrate meal

A history of exercise intolerance, with post-exertional cramps (with or without a family history), suggests a metabolic myopathy, the most common being the glycogen storage disorders. A strong family history and onset in early adulthood suggest a muscular dystrophy. A drug history is important, notably alcohol, which can cause both an inflammatory myopathy and muscle atrophy. Myopathy can be associated with several infections (BOX 1 – 57).

EXAMPLE

The condition started gradually over the preceding one month when the patient had noticed muscle pain and weakness. The patient had noticed that she became unable to ascend stairs or to raise her body from sitting position but her hands function normally. Her muscle weakness was persistent all of the time but got improvement with the use of a course of drugs. The patient had no prior history of drugs intake, infection, or alcohol. Of note the patient developed skin rash around her eye, over her hands, and upper chest.

What is the diagnosis?
The diagnosis is dermatomyositis.

CLINICAL CLUE

LAMBERT EATON SYNDROME
Patients with Lambert Eaton syndrome had antibodies to pre-junctional voltage-gated calcium channels, may have autonomic dysfunction in addition to muscle weakness, but the cardinal clinical sign is absence of tendon reflexes, which can return immediately after sustained contraction of the relevant muscle. The condition is associated with underlying malignancy in a high percentage of cases. These include lung cancer, breast cancer, and ovarian cancer.

LUMP OR MASS
It is not necessarily to imply that appearance of a lump will bring the patient to the hospital. The patients, however, will seek medical advice when there are acute pain, bleeding, color changes, or diameter is increased (ABCD rule).
The common points that should be noted in any patient complain of a swelling are:-

1. The site of the lump.
2. The mode of onset.
3. The characters of the swelling.
4. The relieving factors.
5. The associated features.
6. The timing of the lump (duration and course).
7. The exacerbating and the precipitating factors.
8. The severity of the attack.

SKIN DISEASES

This section is specially designated to fifth and elder medical students. All candidates should know primary and secondary skin diseases (FIGURE 2 – 53). Macule is a flat, colored lesion, < 0.5 cm in diameter, not raised above the surface of the surrounding skin. Patch is a large (> 0.5 cm) flat lesion with a color different from the surrounding skin. This differs from a macule only in size. Nonpalpable purpura is a flat lesion that is due to bleeding into the skin; if <3 mm in diameter, the purpuric lesions are termed petechiae; if >3 mm, they are termed ecchymoses. Palpable purpura is a raised lesion that is due to inflammation of the vessel wall (vasculitis) with subsequent hemorrhage. Papule is a small, solid lesion, <0.5 cm in diameter, raised above the surface of the surrounding skin and hence palpable. Nodule is a larger (0.5–5.0 cm), firm lesion raised above the surface of the surrounding skin. This differs from a papule only in size. Plaque is a large (>1 cm), flat-topped, raised lesion; edges may either be distinct or gradually blend with surrounding skin. Vesicle is a small, fluid-filled papule, <0.5 cm in diameter, raised above the plane of surrounding skin. Pustule is a vesicle filled with pus. Bulla is a fluid-filled, raised, often translucent lesion >0.5 cm in diameter. The bulla may be tense or flaccid and may be on a base of normal or erythematous skin. Wheal is a raised, erythematous, edematous papule or plaque, usually representing short-lived vasodilatation and vasopermeability. Telangiectasia is a dilated, superficial blood vessel. Burrow is a linear or curvilinear papule, caused by a burrowing scabies mite.

Distinguishing primary from secondary lesions is one of the most important parts of the skin examination. Secondary lesions (FIGURE 2 – 54) are modifications, usually made by the patient, of the primary lesion. Lichenification is a distinctive thickening of the skin that is characterized by accentuated skin-fold markings. Scale is an excessive accumulation of stratum corneum. Crust is a dried exudate of body fluids that may be either yellow (i.e., serous crust) or red (i.e., hemorrhagic crust). Verruca is a lesion with papillomatous or warty changes on the surface. Erosion means loss of epidermis without an associated loss of dermis. Ulcer means loss of epidermis and at least a portion of the underlying dermis. Excoriation is linear, angular erosions that may be covered by crust and are caused by scratching. Atrophy is an acquired loss of substance. In the skin, this may appear as a depression with intact epidermis (i.e., loss of dermal or subcutaneous tissue) or as sites of shiny, delicate, wrinkled lesions (i.e., epidermal atrophy). Scar is a change in the skin secondary to trauma or inflammation. Sites may be erythematous, hypopigmented, or hyperpigmented depending on their age or character. Sites on hair-bearing areas may be characterized by destruction of hair follicles.

PRESENTATION: SKIN RASH

HOW TO APPROACH TO SKIN RASH?

S: Site O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Course and Duration) E: Exacerbating and precipitating factors S: Severity

SOCRATES

1. Where is the site (distribution) of the lesion? The distribution of the rash and whether it is symmetrical and universal or asymmetrical should be documented. Symmetrical or universal eruptions usually suggest systemic or constitutional causes while asymmetrical skin rashes suggest dermatological causes.
2. What is the mode of onset? With any rash or skin condition, it is important to determine when it began.
3. What are the characters of the skin lesion e.g. primary or secondary skin lesion?
4. What are the relieving factors? Determine the effects of drugs and sun and cold exposure upon the skin rash
5. What are the associated features? Constitutional symptoms such as fever, headache, fatigue, anorexia and weight loss also need to be documented. Ask if pruritus is associated; localised pruritus is usually due to dermatological disease. Determine if pain or disturbed sensation has occurred; for example, inflammation and edema can produce pain in the skin, while disease involving neurovascular bundles or nerves can produce anesthesia (e.g. leprosy, syphilis). It is important to obtain a past history of rashes or allergic reactions. A past medical history of asthma, eczema or hay fever suggests atopy. Similarly, evidence of systemic disease in the past may be important in a patient with a rash (e.g. diabetes mellitus, connective tissue disease, inflammatory bowel disease). A detailed social history needs to be obtained regarding occupation and hobbies, as chemical exposure and contact with animals or plants can all induce dermatitis. All medications that have been taken must be documented. Orally ingested or parenteral medications can cause a whole host of cutaneous lesions and can mimic many skin diseases. Similarly, a family history of atopic dermatitis, hay fever or skin infestation can be helpful.
6. What is the timing (duration and course) of the rash? The distribution of the rash since noticed, whether it has changed over time, its relationship to sun exposure or heat or cold, and any response to treatment should be documented.
7. What are the exacerbating and the precipitating factors e.g. drugs or heat or cold exposure?
8. What is the severity of the attack?

ANEMIA

This section is especially designated for fifth and elder medical students. Anemia is defined according to the WHO (World Health Organization) as hemoglobin level of less than 13 g/dL in adult men and less than 12 g/dL in adult women. There are three main types of clinical manifestations of anemia. In anemia that has developed rapidly, symptoms related to hypotension may develop as a result of loss of blood volume. In chronic and acute anemias, tissue and organ hypoxia is a major source of symptoms. In hemolytic anemias, there are also specific clinical findings e.g. jaundice. The major factors that determine the specific response of each individual to anemia include the severity and rapidity of the anemia and the patient's overall physical condition. Mild anemia is often associated with no clinical symptoms and may be discovered only when a complete blood cell count is performed for another reason. The earliest clinical symptoms of mild to moderate anemia tend to be a sense of fatigue, generalized weakness, and loss of stamina, followed by tachycardia and exertional dyspnea. In young, healthy patients, these symptoms are not frequently noticed until the hemoglobin level decreases to less than 7 or 8 g/dL. In elderly patients and those with cardiovascular or pulmonary disease, symptoms may occur with even modest degrees of anemia and hemoglobin level of 9 to 11 g/dL. A fourth presentation of anemia is that of hematological malignancy. Features of this presentation may include bone marrow failure (anemia, bleeding, and infection) due to aplastic anemia or leukemia, features of hyperviscosity (nosebleeds, bruising, confusion and visual disturbance) due to multiple myeloma, or classical features (fever, night sweating, and weight loss) of lymphoma. Non-Hodgkin's lymphomas can involve essentially any organ in the body and malfunction of that organ can cause symptoms that lead to the diagnosis. Examples include neurologic symptoms with primary brain lymphoma, shortness of breath with lung lymphomas, epigastric pain and vomiting with gastric ('MALToma') lymphoma, bowel obstruction with small bowel lymphomas, and skin lesions with cutaneous lymphomas. Many lymphomas involve the bone marrow and can occasionally cause bone marrow failure; these patients can present with infections, bleeding, and anemia. Principle causes of anemia are shown in BOX 1 – 58.

BOX 1 – 58. Principle causes of anemia

Decreased or ineffective marrow production	Peripheral causes
Lack of iron, vitamin B_{12} or folate	Blood loss
Aplasia or hypoplasia	Hemolysis
Invasion by malignant cells	Hypersplenism
Anemia of chronic disease	

Hypersplenism means splenomegaly (spleen is rarely enlarged more than 5 cm below the left costal margin in adults, but more marked splenomegaly can occur in childhood and adolescence) with decrease in one or more of blood cells (first thrombocytopenia, then leucopenia, and lastly anemia) despite their active production in the bone marrow (normal bone marrow) and response to splenectomy. Hypersplenism can result from splenomegaly due to any cause. It is commonly seen with splenomegaly due to hematological disorders, portal hypertension, rheumatoid arthritis (Felty's syndrome) and lymphoma.

PRESENTATION: PALLOR OF THE SKIN OR GENERALISED WEAKNESS OR EXERTIONAL SOB

HOW TO APPROACH TO ANEMIA?

SOCRATES

S: Symptoms O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Course and Duration) E: Exacerbating and precipitating factors S: Severity

1. What is the symptom(s) that brought the patient e.g. hypotension as a result of blood loss, symptoms of tissue hypoxia (tiredness, fatigue, or dyspnea), or symptoms of hemolysis (jaundice)? The hemoglobin does not fall immediately with acute severe blood loss, due to proportionate reductions in plasma and red cell volumes (i.e., "people bleed whole blood"). Thus, hemoglobin may be normal or only minimally decreased at the initial presentation of a severe bleeding episode. As extravascular fluid enters the vascular space to restore volume, the hemoglobin falls, but this process may take up to 72 hours. Remember that iron deficiency anemia is the most common type; blood loss is the most common cause of iron deficiency in adults. The loss is usually from the genital tract in women or from the gastrointestinal tract in either sex.
2. What is the mode of onset e.g. acute or chronic? Hemolysis and bleeding present acutely while most other causes of anemia present in more subacute or chronic fashion.

Hemoglobin is a protein specially adapted for gas transport to and from the lungs. It is composed of heme and globin. The metabolism of heme component (iron + biliverdin) had been discussed under the section of jaundice earlier. The globin component is a protein consists of four globin chains that are made from two alpha and two non-alpha chains. Adult hemoglobin consists of hemoglobin A ($\alpha\alpha/\beta\beta$, > 97%), hemoglobin F ($\alpha\alpha/\gamma\gamma$, 1%), and hemoglobin A2 ($\alpha\alpha/\delta\delta$, <2%). Alpha globin chains are produced by two genes on chromosome 16 (thus we have four genes) and beta globin

chains by a single gene on chromosome 11 (thus we have just two genes); imbalance in the production of globin chains produces the thalassemias. Complete absence of beta chain is denoted by β^0 and marked reduction in synthesis is denoted by β^+. Thalassemias are therefore classified in the following way:

1	β/β^+	β-thalassemia minor (trait)
2	β/β^0 or β^+/β^+ or β^+/β^0	β-thalassemia intermediate
3	β^0/β^0	β-thalassemia major
4	- α/αα (one alpha gene is deleted)	α thalassemia carrier
5	- α/- α; - - /αα (two alpha genes are deleted)	α thalassemia trait
6	- α/- - (three alpha genes are deleted)	Hemoglobin H disease
7	- -/- - (four alpha genes are deleted)	Hydrops fetalis

Amino acid substitutions in the beta chains will change the function of the globin chain at critical sites and result in clinical condition called hemoglobinopathies. Examples of hemoglobinopathies are shown here:

1 β6 glutamic acid to valine — Hemoglobin S (sickle)

This means substitution of glutamic acid to valine at position number 6 of the β chain in hemoglobin A

2 β6 glutamic acid to lysine — Hemoglobin C

This means substitution of glutamic acid to lysine at position number 6 of the β chain in hemoglobin A

3 β26 glutamic acid to lysine — Hemoglobin E

This means substitution of glutamic acid to lysine at position number 26 of the β chain in hemoglobin A

Patients may present with combined substitutions in their hemoglobin e.g. HbSC disease or even combined sickle cell anemia and thalassemia. The disease is more severe in patients with HbSS or HbS β^0-thalassaemia than in those with HbS β^+-thalassaemia or HbSC disease.

3. What are the characters of the anemia? Is it due to decreased or ineffective marrow production or due to peripheral causes (see BOX 1 – 58). In this field one should ask about relevant occupational, medical, surgical, menstrual, dietary, and family history. Occupational history may provide a clue to the underlying cause; painting may cause lead poisoning that leads to hemolytic anemia, exposure to benzene in industry (or radiation exposure as with radiologists) may lead to leukemia, and exposure to insecticides may lead aplastic anemia; medical history may reveal a disease which is known to be associated with anemia, such as rheumatoid arthritis (the anemia of chronic disease), celiac disease (iron and folate deficiency), and Crohn's (iron and vitamin B_{12}); surgery involving the stomach may lead to iron and vitamin B_{12} deficiency anemia, surgery involving the upper small bowel may lead to folate deficiency, and surgery in the terminal ileum may lead to vitamin B_{12}. Hemolytic anemias such as the hemoglobinopathies and hereditary spherocytosis may be suspected from the family history. Pernicious anemia may also be familial. A dietary history (BOX 1 – 59) should assess the intake of iron, folate, and vitamin B_{12} which may become deficient in comparison to needs e.g. in pregnancy or during periods of rapid growth.

BOX 1 – 59. Dietary assessment

Diet rich in iron	Meat and fish
Diet rich in vitamin B_{12}	Meat, fish, eggs and milk
Diet rich in folate	Leafy vegetables (spinach, broccoli, lettuce), fruits (bananas, melons) and animal protein (liver, kidney)

4. What are the relieving factors (the treatment) of anemia e.g. drugs like hematinics (iron, folate, cobalamin), hydroxyurea (used in sickle cell anemia), steroids (BOX 1 – 60), danazole (used in idiopathic thrombocytopenia purpura and in myelofibrosis), or chemotherapy and radiotherapy (used in leukemias, lymphomas, and multiple myeloma) or blood transfusion? Ask also about the number of blood pints that the patient received.

BOX 1 – 60. Steroid uses in hematological diseases

1	Autoimmune hemolytic anemia
2	Idiopathic thrombocytopenia purpura (ITP)
3	Aplastic anemia
4	Primary myelofibrosis
5	Lymphomas
6	Acute leukemia
7	Multiple myeloma

Other modalities used in the treatment of hematological disorders may include plasma exchange procedure (plasmapheresis) or splenectomy. The general indications for both are provided in BOX 1 – 61.

BOX 1 – 61. General indications of splenectomy and plasma exchange

General indications of splenectomy	General indications of plasma exchange (plasmapheresis)
1. Massive enlargement that causes pain and hypersplenism • Thalassemia • Hereditary spherocytosis • Chronic idiopathic thrombocytopenia purpura (ITP) • Hairy cell leukemia • Warm autoimmune hemolytic anemia 2. Staging in Hodgkin's disease 3. Trauma to the spleen	Hyperviscosity syndrome e.g. secondary to multiple myeloma Guillain Barré syndrome Myasthenia gravis Goodpasture's syndrome ANCA positive vasculitis e.g. Churg – Strauss syndrome Thrombotic thrombocytopenia purpura (TTP) Hemolytic uremic syndrome (HUS) Cryoglobulinemia

5. What are the associated factors e.g. tiredness, jaundice, gastrointestinal bleeding, or heavy menstruation? Iron deficiency anemia is the most common type of anemia world-wide. A thorough gastrointestinal history is important, looking in particular for symptoms indicating blood loss. Menorrhagia is a common cause of anemia in females still menstruating, so women should always be asked about their periods. In addition women should be asked about pregnancy and breast feeding. Patients with hematological malignancy may present with a variety of clinical pictures (see above).

   A clinical manifestation unique to iron deficiency is pica, which is an unusual craving for certain non-nutritional substances. Pica may be manifested as a craving for ice (pagophagia), or less commonly for clay (geophagia) or starch (amylophagia); pagophagia is believed to be the most specific to iron deficiency.
   When iron deficiency is severe and chronic, widespread tissue changes may be present, including koilonychia, angular stomatitis, glossitis, hair thinning, and pharyngeal webs (Paterson–Kelly syndrome).

6. What is the timing of anemia (duration and course)? Ask about the time of anemia and the course since noted. For example, thalassemia major becomes symptomatic as severe and progressive hemolytic anemia during the second six months of life. Such patients present with a long history of blood transfusion and chelating agent e.g. subcutaneous deferoxamine or oral deferasirox. Sickle cell disease may also become noticeable in the first year of life. Such patients may present with recurrent attacks of crises. Both Fanconi's aplastic anemia and acute lymphocytic anemia may present early in life. The patients should also be asked about radiotherapy or chemotherapy (BOX 1 – 62) if he/she receive.

BOX 1 – 62. Some examples of chemotherapeutic agents

Alkalating agents	
Cyclophosphamide	Marrow suppression, hemorrhagic cystitis, transitional cell carcinoma, and common alkylators
Chlorambucil	Common alkylators
Cisplatin	Ototoxicity, peripheral neuropathy, and hypomagnesemia
Antitumor antibiotics	
Bleomycin	Pulmonary fibrosis, skin effects, Raynaud's phenomenon, and hypersensitivity reaction
Doxorubicin and daunorubicin	Marrow suppression, mucositis, alopecia, and cardiovascular reaction (acute/chronic). Cardiac toxicity is increased when dose > 550 mg/m^2 and is irreversible
Antimetabolites	
Azathioprine	Marrow suppression, hepatic fibrosis, and nausea
Methotrexate	Marrow suppression, folate antagonist, hepatic fibrosis, and pneumonitis
Hydroxyurea	Marrow suppression, mucositis, skin changes, and rarely renal, liver, lung, and CNS toxicity
5-Fluorouracil	Marrow suppression, mucositis, skin changes, and neurologic toxicity
Antimitotic agents	
Vincristine	Marrow suppression, neuropathy, ileus, constipation, bladder toxicity, SIADH, and CVS toxicity
Vinblastine	Marrow suppression, neuropathy (less common), hypertension, and Raynaud's phenomenon

Common alkylator causes alopecia, pulmonary, infertility toxicity, plus teratogenesis

7. What are the exacerbating and precipitating factors? Fava bean induces hemolysis in G6PD deficiency. Anemia may also be precipitated by ingestion of drugs (BOX 1 – 63)?

BOX 1 – 63. Drugs causing anemia	
Iron deficiency anemia	Proton pump inhibitor and NSAID
Vitamin B_{12} deficiency anemia	Metformin
Folate deficiency anemia	Certain anticonvulsants (e.g. phenytoin) and contraceptive pills
Sideroblastic anemia	Anti-tuberculous drugs e.g. isoniazid and chloramphenicol
Hemolysis in G6PD deficiency	Aspirin, sulphonamides, nitrofurantoin, ciprofloxacin, vitamin K, and dapsone
Warm autoimmune hemolysis	Methyldopa, mefenamic acid, penicillin, quinine
Aplastic anemia	Chloramphenicol, sulphonamides, antithyroid drugs, and anticonvulsants
Myelofibrosis	Calcium
Note that anti-cancer drugs may produce any of the above mentioned types of anemia	

8. What is the severity of anemia? Severity is determined as to whether anemia interferes with the patient's daily activities and necessitates blood transfusion (BOX 1 – 64).

BOX 1 – 64. Indications of blood transfusion in anemia
Severe hemorrhage or hemolysis
Patients who develop angina, heart failure or evidence of cerebral hypoxia due to anemia
Very low hemoglobin

EXAMPLES

The condition started as exertional SOB that occurred gradually over two to three months, slightly relieved by rest and blood transfusion, and aggravated by exertion. She had one similar attack one year ago for which she received two pints of blood and resulted in complete improvement in her condition. She had failed two and half years ago to respond to iron course of therapy when she felt at that time generalised fatigue and weakness. Six months later she had been subjected to further investigation and given daily oral folate tablet after two pints of blood. She however neglected her medication for the preceding four to five months in a mistaken belief that she had cured. She had healthy diet, no drugs ingested prior to this attack and no significant relevant medical illness. Both her father and mother had a blood problem.

What is the diagnosis?
The diagnosis is hemoglobinopathy e.g. thalassemia.

The condition started over the preceding three days as recurrent attacks of bone pain that was located mainly in the upper part of his arm, mild at the start but soon became very severe likened trauma pain, dull ache in nature, similar to previous two attack in this year, relieved slightly by rest and pain medication, and aggravated by movement. He had two similar attacks in the current year each persisted for two to three weeks in a waxing and waning pattern. The patient had been kept on daily folate therapy when he developed exertional SOB two years ago and received two pints of blood. His condition was associated with generalised weakness, high-grade intermittent fever with sweating, and occasional awareness of his beats. He had otherwise healthy diet, no drugs ingested prior to this attack and no significant relevant medical illness. Both his father and mother had a blood problem.

What is the diagnosis?
The diagnosis is vaso-occlusive crisis in sickle cell anemia.

SYSTEMIC BLEEDING

This section is especially designated for fifth and elder medical students. Hemostasis depends upon interactions between the vessel wall, platelets and clotting factors. There are two phases of hemostasis: primary and secondary. In the primary phase, the damaged vessel constricts and platelets aggregate at the site of damage to form a plug to arrest hemorrhage within a few minutes. This is followed by activation of the coagulation system with secondary deposition of a fibrin mesh to secure the platelet plug. Bleeding therefore results from disorders affecting the vessel wall, the clotting factors, the platelets, combination of them, or Von Willebrand syndrome (BOX 1 – 65).

BOX 1 – 65. Causes of systemic bleeding

DECREASE IN THE PLATELET NUMBER (THROMBOCYTOPENIA)		
1	Hereditary	Wiskott-Aldrich syndrome. These patients usually have a triad of significant thrombocytopenia with small platelets noted on the peripheral smear, congenital immunodeficiency, and eczema
2	Acquired	Drug-induced thrombocytopenia e.g. sulfonamides, thiazides, heparin Idiopathic thrombocytopenia purpura (primary and secondary ITP) Infection-induced thrombocytopenia e.g. EBV, HIV, and Gram-negative septicaemia Increased consumption of the platelets e.g. DIC, TTP, HUS Marrow hypoplasia or aplasia e.g. aplastic anemia (primary and secondary) Marrow infiltration e.g. leukemia, myeloma, carcinoma, myelofibrosis Lack of vitamin B_{12}/folate
DECREASE IN THE PLATELET FUNCTION (THROMBASTHENIA)		
1	Hereditary	Bernard-Soulier syndrome and Glanzmann's thrombasthenia
2	Acquired	Drug-induced thrombasthenia e.g. anti-platelets, NSAID, β –blockers Uremia
DECREASE IN THE CLOTTING FACTORS PRODUCTION		
1	Hereditary	Hereditary hemophilias e.g. hemophilia A or hemophilia B
2	Acquired	Acquired hemophilias. Autoantibody inhibitors can occur spontaneously in individuals with previously normal hemostasis (nonhemophiliacs). Although approximately 50% of patients have no obvious underlying cause, the remainder of cases are associated with autoimmune diseases, lymphoproliferative disorders, idiosyncratic drug associations, and pregnancy Drugs e.g. warfarin
VESSEL WALL DISORDERS		
1	Hereditary	Hereditary hemorrhagic telangiectasia and Ehlers-Danlos disease
2	Acquired	Trauma Drugs e.g. corticosteroids
COMBINED DEFECTS		
1	Liver disease	Reduced hepatic synthesis of clotting factors and fibrinogen Reduced clearance of plasminogen activator Thrombocytopenia secondary to hypersplenism Cholestatic jaundice reduces vitamin K absorption
2	Renal failure	Both thrombocytopenia and thrombasthenia
VON WILLEBRAND DISEASE		

A history of bleeding is often remarkably reproducible, particularly after dental extraction; if a socket oozes for 2 days after removal of a tooth on one occasion; this is likely to recur following each subsequent extraction.

PRESENTATION: BLEEDING

HOW TO APPROACH TO SYSTEMIC BLEEDING?

SOCRATES

S: Site O: Onset C: Character R: Relieving factors A: Associated factors
T: Timing (Course and Duration) E: Exacerbating and precipitating factors S: Severity

1. Where is the site of bleeds? Does the attack involve single or multiple sites? Muscle and joint bleeds indicate a coagulation defect, whereas purpura, petechiae, ecchymoses, prolonged bleeding from superficial cuts, epistaxis, gastrointestinal hemorrhage or menorrhagia indicates a thrombocytopenia, thrombasthenia, or von Willebrand disease. Bleeding at a single site suggest a local structural abnormality where as multiple sites bleeding suggests coagulopathy. However, even diffuse bleeding may arise from structural rather than hemostatic abnormalities (e.g., recurrent mucosal hemorrhage in patients with hereditary hemorrhagic telangiectasia). Conversely, a single episode of bleeding from an isolated site may be the initial manifestation of a systemic coagulopathy.
2. What is the mode of onset e.g. acute or chronic illness?
3. What is the character of bleeding e.g. gastrointestinal bleeding, vaginal bleeding?
4. What are the relieving factors e.g. drugs, blood transfusion (BOX 1 – 66), platelet transfusion, clotting factors infusion?

BOX 1 – 66. Blood and blood products

1	Whole blood	450 ml donor blood collected into 63 ml anticoagulant/preservative solution. Shelf life up to 5 weeks. It is given as 20 cc/kg or by noting that each pint of blood increases hemoglobin 1 g/dL
2	Red cell concentrate	Most of the plasma removed and replaced with a solution to optimise preservation of red cells. Each pint increases hemoglobin 1 g/dL
3	Platelet concentrate	One adult dose is made from four or five donations of whole blood, or from a single platelet apheresis. Shelf life up to 5 days from collection. Each pint increases platelets 5000 – 10000
4	Plasma (Fresh frozen plasma)	150-300 ml plasma obtained from one donation of whole blood. Shelf life is usually 1 year. It is given as 15 cc/kg or by noting that each pint increases clotting factors 30%
5	Cryoprecipitate	Each 10-20 ml pack of precipitate contains fibrinogen, factor VIII and von Willebrand factor. It is given as 1 unit (bag) for each 5 kg
6	Factor VIII concentrate	Factor VIII is given according to the following equation VIII dose (IU) = Target FVIII levels – FVIII baseline levels x body weight (kg) x 0.5 unit/kg
7	Factor IX concentrate	Factor IX is given according to the following equation FIX dose (IU) = Target FIX levels – FIX baseline levels x body weight (kg) x 1.0 unit/kg

5. What are the associated features e.g. pallor, fever, or jaundice?
6. What is the timing of the attacks (duration and course)? It may be possible to assess whether the disorder is congenital or acquired.
7. What are the exacerbating and precipitating factors e.g. drugs, family history, medical history, and surgical history? Absence of relatives with clinically significant bleeding does not exclude a hereditary bleeding diathesis; about one-third of cases of hemophilia arise in individuals without a family history. Many diseases (or their treatment) may be associated with bleeding but hepatic or renal failure, DIC, paraproteinemia or a connective tissue disease in particular should be considered. Ask about all operations. Dental extractions, circumcision, tonsillectomy, labor and delivery, menses, vaccinations, and injections are particularly stressful tests of the hemostatic system. Bleeding that starts immediately after surgery indicates defective platelet plug formation and primary hemostasis, whereas that which comes on after several hours suggests a coagulation defect.
8. What is the severity of the attack? Bleeding arising spontaneously indicates a more severe defect than bleeding that occurs only after trauma.

EXAMPLES

The condition started yesterday as a tingling and burning sensation in the right knee joint before the onset of moderately severe joint pain and limitation of joint movement. The attack started when the patient had exposed to minor trauma during football playing then the knee joint became hot, red, swollen, and restricted in movement. The patient had previous similar episodes that some of them started spontaneously whilst other started after trauma. The patient experienced that he became better after rest and replacement therapy as his condition started at the age of 18 months. His condition was associated with high-grade intermittent fever and sweating. He had otherwise healthy diet, no drugs ingested prior to this attack and no significant relevant medical illness. Both his father and mother had no similar problem.

What is the diagnosis?
The diagnosis is hemophilia.

The condition started insidiously over the preceding one week as attacks of nasal bleeding that was followed by heavy non-menstruating vaginal bleeding. The patient in addition had experienced easy bruising of her skin. The bleeding was not trauma-induced and replaced by proper blood components but continued despite these measures. The patient had no previous similar episodes, no drugs intake, no relevant medical illness, and both her parents are healthy and alive.

What is the diagnosis?
The diagnosis is thrombocytopenia probably idiopathic thrombocytopenia purpura.

LEARNING POINTS IN THE HISTORY OF PRESENT ILLNESS

1. Medical terms e.g. diarrhea and dyspnoea should be discouraged and instead use frequent bowel motions, shortness of breath (SOB) unless the word is popular as well as medical e.g. cough, vomiting, headache, or fever since these words are the patient's own wording.
2. Try not to mention names of doctors, instead use the following terms that are regarded as a source of referral:-
   - Private clinic.
   - Inpatient clinic.
   - Primary health center.
   - Hospital.
3. Any prior diagnosis in the history of present illness should not be recorded whether an informant said that or a patient's doctor unless the patient is a known case for that disease e.g. diabetes mellitus, hypertension, ischemic heart disease, epilepsy and bleeding tendency and the current history is belonging to it. Here, the history of present illness is started by the following sentence (the patient is a known case of..... since .....). An example for that is the following case: a patient who had ischemic heart disease for 3 years duration, 2 days earlier, he developed chest pain. Thus the history of the present illness will be like this: the patient is a known case of ischemic heart disease since 2010, two days prior to admission he developed chest pain that was.....
4. The patient's complain should thoroughly be analyzed together with the associated symptoms. The remaining symptoms of the system involved should also be analyzed but to a lesser extent. An example for that is when a patient presented with abdominal pain. This pain should be thoroughly analyzed then the associated symptom(s) e.g. vomiting is analyzed. Lastly the related remaining symptoms of the gastrointestinal tract e.g. constipation because the patient's complain was one of the gastrointestinal symptoms.
5. In case of surgical history, determine whether the patient is post operative or not. If postoperative, determine in which post operative day the patient is. Day zero is the day of operation while day one is the day after the operation. Here, ask about the fluid and medications that the patient received up to the time of history taken. Also ask about bowel motion and patient's movement.
6. If the patient for example suffered from headache for 3 days prior to admission, but developed abdominal pain for 1 day later that brought him/her to the hospital. Thus the chief complaint is abdominal pain for 2 days prior to admission, but the history of present illness will be as follow (the condition started as headache that was ....., one day after that the patient suffered from abdominal pain that was .....).
7. If the chief complaint for example is chest pain or dyspnoea so the systems involved are the cardiovascular and respiratory systems that should analyzed in the history of present illness and are not mentioned in the next step of history which is review of other systems.
8. Patients with metabolic disease e.g. chronic renal failure when develop decompensated event they usually present with shortness of breath (SOB) due to fluid overload. The systems involved are thus the respiratory and the cardiovascular systems. In such exceptional case or any similar case one should in addition analyze the genuine underlying system which is the urolological system looking particular at the urine volume, color, and form or edema in the history of present illness. A similar case involves the hematological system when a patient presented with shortness of breath (SOB) due to anemia.
9. During analysis of the case, you may use common abbreviation like HTN, DM, or IHD. Try not to orally present them as most of them are non universal abbreviations. Instead present these data as hypertension, diabetes mellitus, or ischemic heart diseases.
10. With practice you will develop the capability of avoiding written presentation as this manner lack the clinical experience and make some examiner unsatisfied. The best way is to ordinarily asking the patient, writing the information, repeating them with your self, reiterating them to the patient to confirm or to correct, and finally presenting the case orally to the examiners without case sheath.
11. Try to make a history containing even minor events. This is especially applied for undergraduate students as they may have not the capability to separate which is important and which is trivial. Once the story of the patient is complete, try to ask about the following:-
    - Admission whether to the emergency unit, intensive care unit, to the ward or the patient consulted a private clinic then admission was advised.
    - Basic investigation that the patient performed e.g. routine blood sampling, general urine examination, general stool examination, ultrasonography, chest X-ray or ECG. Each case should of course individualize.
    - Medications that the patient received.
    - Improvement or worsening in the patient's condition now. Here, you must ask about type and time of that symptom improved or worsened.
12. Postgraduate students may need in addition to recollect the history into a short medical story containing the medically correct terms. For example a 70-year-old male patient presented with a three days history of dyspnoea, chest pain and fever that then progressively worsened till the time of his admission as a confused case.

3. REVIEW OF OTHER SYSTEMS
This part includes leadingly directed questions for each system not involved in the history of present illness:

NERVOUS SYSTEM (MNEMONIC: SHOVE + FEVER)
- Syncope, speech defect, swallowing difficulty.
- Headache, history of motor or sensory disturbances including paresthesia, vision, hearing, and smell changes as well as tremor, incoordination, or weakness. Loss of consciousness and changes of behavior or psyche.
- Ocular disturbance including diplopia and field defect.
- Vertigo and dizziness.
- Epilepsy; seizure.
- Fever.

CARDIOVASCULAR SYSTEM
Dyspnoea, orthopnoea, paroxysmal nocturnal dyspnea, syncope, palpitation, chest pain, ankle edema, cyanosis, and symptoms of peripheral arterial diseases (intermittent claudication, rest (night) limb pain and tissue loss).

RESPIRATORY SYSTEM (CC CHWSH)
- Chest pain and cyanosis.
- Cough, hoarseness of voice, wheezing, sputum, and hemoptysis.
- Dyspnoea derivatives; dyspnoea, orthopnoea, trepopnoea, platypnoea, apnoea.

GASTROINTESTINAL TRACT (GIT)
- Weight loss, increase or decrease in appetite, alteration in taste, water brush, dry mouth.
- Nausea, vomiting, regurgitation, and rumination.
- Heartburn and indigestion.
- Aphagia, globus pharangeus, dysphagia, odynophagia, and phagophagia.
- Hematemesis, malena, and rectal bleeding.
- Abdominal pain or distension.
- Diarrhea, constipation, or tenesmus.
- Jaundice.

UROLOGICAL SYSTEM
- Frequency, urgency, dysuria, (remember UTI).
- Loin pain and hematuria (remember stone).
- Hesitancy, terminal dribbling, retention, and incontinence (remember benign prostatic hyperplasia).
- Change in the color, form (foamy), or odor of urine.
- Polyuria, oliguria, annuria, and nucturia (volume).
- Pneumaturia.
- Edema of the ankles, hands or face.

MUSCULOSKELETAL SYSTEM
Muscle weakness pain or stiffness, joint pain, swelling, or limitation of joint movement, and bone pain (including back).

INTEGUMENTARY SYSTEM
- Change in the color of skin, skin rash, and skin itching.
- Skin accessories; nail changes, hair loss or hirsutism, and oral/genital ulceration.

HEMATOLOGICAL SYSTEM
Anemia, bleeding, thrombosis, past transfusions and/or transfusion reactions.

ENDOCRINE SYSTEM
Fatigue, thyroid trouble (for hypothyroidism ask about tiredness and weakness; for hyperthyroidism ask about hyperactivity and irritability) and heat or cold intolerance, excessive sweating and excessive thirst or hunger, altered facial appearance or change in glove or shoe size, weight gain or weight loss, hirsutism, galactorrhea, gynecomastia, flushing, and erectile dysfunction/loss of libido

4. PAST MEDICAL HISTORY
This part of history is specialized for medical problems that are neither related to the current illness nor belonging to it. For example a patient who is suffering from melena as he has duodenal ulcer so duodenal ulcer is not a past medical history, but the same patient may have epilepsy that is probably (although may) unrelated directly to melena so epilepsy is a past medical history. In general inquiring should be directed towards the following medical illnesses:-
- Diabetes mellitus

- Hypertension
- Asthma
- Epilepsy
- Bleeding tendency
- Myocardial infarction
- Heart failure
- Chronic renal disease

5. PAST SURGICAL HISTORY

Past surgical history should include all important operations from infancy onward that are unrelated to or belonging to the presenting illness. It is noteworthy to record circumcision in males and tooth extraction in both males and females (if the case is related e.g. bleeding). It is wise to ask about the operation in the following manner:

- Cause of operation
- Type of operation
- Time of operation

6. MENSTRUAL AND GYNECOLOGICAL HISTORY

In this part of the history ask about the following:

- G P A where G is Gravida which means number of pregnancies regardless its outcome, P is Para which means number of deliveries after 28 weeks of gestation regardless a fetus live or dead and A is abortion which means number of fetal loss before 24 weeks of gestation.
- Date of menarche or menopause. Menarche is the first menstruation usually between 11-14 years while menopause refers to the time of cessation of periods.
- Regularity of the cycle.
- Dysmenorrhoea.
- Usual duration of each period and length of cycle, counting from the first day of one period to the first day of the next. This information is generally recorded as (M.C. 12 4/28 regular) meaning the periods began at the age of 12, last 4 days and occur every 28 days.

CLINICAL CLUE

It is advisable to ask young patient about menarche, but in elder female ask about menopause.

7. DRUGS AND HABIT HISTORY

DRUG HISTORY

Ask the patient whether he or she is taking any drug; the patient will often describe these by color or size rather than by name and dose. Note the name, dose, length of use, and the indication for each drug. Remember that some drugs are prescribed as transdermal patches or subcutaneous implants. Always ask specifically whether a woman is taking the contraceptive pill, because this is not considered a medicine or tablet by many who take it. The same is true of inhalers. To remind the patient, it is often useful to ask about the use of classes of drugs. A basic list should include questions about treatment for blood pressure, high cholesterol, diabetes, arthritis, impotence, contraception, epilepsy, anticoagulation and the use of antibiotics. Also ask the patient if he or she is taking any over-the-counter preparations (e.g. aspirin, antihistamines, vitamins). Aspirin and standard non-steroidal anti-inflammatory drugs (NSAIDs), but not paracetamol, can cause gastrointestinal bleeding. Patients with chronic pain may consume large amounts of analgesics, including drugs containing opioids such as codeine or morphine. A careful history of the period of use of opioids and the quantities used is important because they are drugs of dependence. There may be some medications or treatments the patient has had in the past which remain relevant. These include corticosteroids, chemotherapeutic agents (anti-cancer drugs) and radiotherapy. Often, patients, especially those with a chronic disease, are very well informed about their condition and their treatment. Note any adverse reactions in the past. Also ask about any allergy to drugs (often a skin reaction or episode of bronchospasm) and what the allergic reaction actually involved. Often the patient confuses an allergy with a side-effect of a drug. Remember that not all medical problems are treated with drugs. Ask about courses of physiotherapy or rehabilitation for musculoskeletal problems. Certain gastrointestinal conditions are treated with dietary supplements (e.g. pancreatic enzymes for chronic pancreatitis) or restrictions (e.g. of gluten for coeliac disease).

ALCOHOL INTAKE

Ask whether the patient drinks alcohol or is teetotal. If so, ask about the type, the amount and the frequency. If the patient claims to be a social drinker, find out exactly what this means. It is useful in estimating alcohol consumption to understand that one beer, four ounces of wine, or one ounce of 80% spirits all contain ~12 g of alcohol. The threshold

for developing alcoholic liver disease in men is an intake of >60-80 g/d of alcohol for 10 years, while women are at increased risk for developing similar degrees of liver injury by consuming 20–40 g/d. Individuals who consume large quantities (>90 g/d) of alcohol over many years may develop dilated cardiomyopathy (DCM). In the UK, the current recommended safe limits are 28 units (of ethanol) a week for men and 21 units (of ethanol) for women. Certain questions can be helpful in making a diagnosis of alcoholism; these are referred to as the **CAGE** questions:

- Have you ever felt you ought to **C**ut down on your drinking?
- Have people **A**nnoyed you by criticising your drinking?
- Have you every felt bad or **G**uilty about your drinking?
- Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover ('**E**ye opener')?

A 'yes' to any of these questions suggests that there may be a serious drinking problem, and further inquiry into the history of this is important. Ask the patient about the pattern (daily/weekly), usual place of drinking, and whether the patient drinks alone or accompanied.

CLINICAL CLUE

Remember that approximately each unit of ethanol is equal to 8 grams.

TOBACCO SMOKING

Cigarette smoking is a risk factor for vascular disease, chronic lung disease, several cancers and peptic ulceration, and may damage the fetus. Cigar and pipe smokers typically inhale less smoke than cigarette smokers, and overall mortality rates are correspondingly lower in this group, except from carcinoma of the oral cavity, larynx and oesophagus. It is important to determine whether the patient is a smoker, an ex-smoker, passive (second hand smoker) or a life long non-smoker. If the patient is smoker, determine the form (cigars, cigarettes, narghile (hubble-bubble) or pipe), the amount (number of cigarettes, cigars, or amount of pipe/narghile tobacco per day), and the duration since first start smoking. If the patient is an ex-smoker, the length of time since the practice ceased should be noted as some patients may claim to be a non-smoker if he or she stopped smoking that morning.

DRUG ABUSE

Ask about 'recreational' drug use. The use of intravenous drugs has many implications for the patient's health. Ask whether any attempt has been made to avoid sharing needles. This may protect against the injection of viruses, but not against bacterial infection from the use of impure substances. Patients tend to be defensive and are quite likely to deny or minimize the smoking, drinking or drug abusing; therefore, enquiring requires training and experience

8. SOCIOECONOMIC HISTORY

This part of history is applied to determine the socioeconomic status of the patient. It is generally inquired via the following axes of questions:

- Type of home, size-owned or rented
- Rural or urban area
- Water supply (purified water, tap water, well water, or river water)
- Indoor or outdoor domestic animals

9. FAMILY HISTORY

In this part of the history determine the cause of death of the patient's first degree relative e.g. parents, brothers and sisters and the presence of any heritable disease e.g. diabetes mellitus or hypertension.

10. HISTORY OF HOSPITALIZATION

This part of history is concerning with hospitalization history seemingly not related to any of the pre-mentioned parts of history for example road traffic accidents (RTA).

CLINICAL CASES

The following cases are real cases; only the personal history is modified for medicolegal purpose.

CASE I CASE PRESENTATION IN INTERNAL MEDICINE/CARDIAC CASE

Mr. Ali Omer Josef Al-Zibari who is 55 years old, living in Al-Amel quarter/Karbala, Moslem, married and had 5 daughters and 5 sons. He is a baker. His next of kin is Fatima M. Ali Al-Zibari; his wife. The patient was admitted into the hospital on 5th of September 2004 chiefly complaining from chest pain for 3 hours prior to admission.

Mr /mɪstə(r)/is a short form of Mister e.g. Mr Ahmed and used in front of the name of a man when talking politely to him or about him. Ms /məz/ is a short form of Miss e.g. Ms Lyla and used as a formal title in front of the name of a woman who is not married when talking to her or about her. Mrs /mɪsɪz/ is a short form of Mistress e.g. Mrs Indiana and used a formal title in front of the name of a woman who is married when talking to her or about her.

Regarding history of present illness, the patient is a known case of hypertension since 2001. Three weeks prior to admission he developed SOB that was mild at the start, experienced only on walking for along distance about 300 meters, but gradually built up to be experienced on lesser distance then even on lying flat, relieved by sitting and head up sleeping, associated with intermittent productive cough occurring mainly at night to be then experienced all over the day, of copious amount (more than cupful per day), mucopurulent sputum that sooner became purulent and contained streaks of blood. After that i.e. 3 hours prior to admission, the patient developed sudden chest pain of sever degree that interfered with the patient's daily activities such as eating or sleeping, lasting for about 20 minutes, of constricting character that is not relieved by resting or sublingual medication during the attack, radiated to the neck and left arm, aggravated by any movement from the patient, and associated with brief period of awareness of heart beats. After these 20 minutes, the chest pain slightly settled thus the patient neglected him self to be recur again, associated with nausea and high grade fever as well as attack of severe SOB that made the patient discolored blue as well as sweaty. Thus the family consulted a private clinic and admission was advised to the patient. Of note the patient had not briefly lost his consciousness during the attack and there were no experience of ankle edema, pain in calf on walking, or rest (night) pain. Upon admission, the patient was immediately brought to the coronary care unit where he received oxygenation and intravenous medication. An ECG, chest x-ray and blood sampling for routine examination were also performed. Now the patient feels better where the chest pain had been improved, temperature had been decreased, but mild degree of SOB is still present.

Regarding review of other systems

In the nervous system, there was no syncope, no speech defect, no swallowing difficulty, no headache, and no history of motor or sensory disturbances. No loss of consciousness or changes of behavior or psyche. No ocular disturbance, no vertigo, and no seizure.

In the GIT symptoms, there was poor appetite for the previous 3 days. No weight loss, no water brush, and no dry mouth. No nausea, vomiting, regurgitation, or rumination. No heartburn or indigestion. No dysphagia or odynophagia, no hematemesis, melena or rectal bleeding, no abdominal pain or distension, no diarrhea or constipation, and no jaundice.

In the urological system, there was no frequency, urgency, or dysuria. No loin pain or hematuria, no hesitancy, terminal dribbling, retention or incontinence. No change in urine color, form, volume or odor.

In the musculoskeletal system, no muscle weakness pain or stiffness, no joint pain, swelling, or limitation of joint movement, and no bone pain.

In the intugumentary system, no change in the color of skin, skin rash, or skin itching. No nail changes, hair loss or hirsutism, and no oral/genital ulceration.

Remember that both cardiovascular system and respiratory system had not mentioned in review of other systems as both being analyzed in the history of present illness.

Regarding past medical history, the patient is hypertensive since 2001, diagnosed on the basis of three high blood pressure readings despite trial of no added salt diet with regular physical activity. No change in his medication since that time and no adverse effect had the patient noticed. He had no diabetes mellitus, no asthma, and no bleeding tendency.
Regarding past surgical history, the patient had not performed any operation.
Regarding drug and habit history, the patient is on chronic use of diltiazem. He has no allergy to penicillin or sulfa-containing drug. He is smoker; about 30 cigarettes/day since 1970, but neither alcoholic nor addict.
Regarding socioeconomic history, the patient is living in urban area in an owned home; purified water is the water supply, no indoor or outdoor domestic animals.
Regarding family history, patient's father died because of heart failure and his mother also died because of myocardial infarction. Among his brothers, he had a positive family history for ischemic heart disease.

Note that the menstrual and gynecological history had been neglected as the patient is male.

CASE II CASE PRESENTATION IN GENERAL SURGERY

Ms. Sarah Ali Hussein Aljubori; she is 15 years old, living in Baghdad/Al-Amel quarter, Moslem, unmarried, and is a student in the third intermediate school. Her next of kin is her older sister; Amerah A. H. Aljubori. The patient is admitted into the hospital on twelfth of February/2005.
Regarding the chief complain and its duration, the patient suffered from abdominal pain for 18 hours.
Regarding history of present illness, the condition started as an abdominal pain that was of a sudden onset, localized in the upper central part of the abdomen then shifted to the lower right part experienced at 2 a.m., lasting for 2 hours then gradually decreased in intensity. It was of a severe degree, colicky in character, interfering with the patient's daily activities as sleeping or eating, aggravated by walking and slightly relieved by rest, associated with yellow colored, bitter tasted, little contents vomiting about 3 to 4 times that preceded by nausea. The patient also experienced an inability to pass stool during the time of the attack as well as frontally localized headache and slight increased body temperature. Thus the patient consulted a private clinic where admission was advised to her and upon admission, an ultrasonography as well as blood sampling for routine check up were done and immediately then candidates for operation. The patient now is on day one post operatively where she received i.v. fluid, injectable forms of medication and she can pass motion and move gradually.
Regarding review of other systems:-
In the nervous system, there was no syncope, no speech defect, no swallowing difficulty, no headache, and no history of motor or sensory disturbances. No loss of consciousness or changes of behavior or psyche. No ocular disturbance, no vertigo, and no seizure.
In the cardiovascular system, there was no dyspnoea, no PND, no orthopnoea, no platypnoea, no palpitation, no chest pain, no ankle edema or cyanosis.
In the respiratory system, there was no cough, no sputum, no hemoptysis, no hoarseness of voice and no wheezing.
In the urological system, there was no frequency, urgency, or dysuria. No loin pain or hematuria, no hesitancy, terminal dribbling, retention, or incontinence. No change in the color, form, volume or odor of urine.
In the musculoskeletal system, no muscle weakness pain or stiffness, no joint pain, swelling, or limitation of joint movement, and no bone pain.

In the intugumentary system, no change in the color of skin, skin rash, or skin itching. No nail changes, hair loss or hirsutism, and no oral/genital ulceration.
Regarding past medical history, the patient had a history of extrinsic asthma; for which she is kept on as required salbutamol inhalers since 2000. She had no diabetes mellitus, no hypertension, and no bleeding tendency.
Regarding past surgical history, operation was performed to her in the form of splenectomy 3 years age due to trauma to the abdomen.
Regarding menstrual and gynecological history, she is Gravida nil, Para nil (M.C. 13 5/28 regular) Her periods began at the age of 13 years, regular cycle, occur every 28 days, last 5 days, and not associated with dysmenorrhea
Regarding drug history, she is on chronic use of as required salbutamol inhalers; no adverse effects had been encountered. No allergy to penicillin or sulfa-containing drug. She is not smoker, neither alcoholic nor addict.

Ask the patient whether she received medication in the form of oral capsule for any attacks of pharyngitis or tonsillitis to clarify penicillin allergy when she/he does not know about penicillin allergy. Also ask the patient whether she received medication in the form of oral tablet for UTI to clarify sulfa-containing drugs allergy.

Regarding socioeconomic history, the patient is living in urban area; purified water is the source of water; no indoor or outdoor domestic animals.
Regarding family history, both father and mother are alive; no heritable diseases.

CASE III CASE PRESENTATION IN INTERNAL MEDICINE/NEUROLOGICAL CASE

Mrs. Salma Uda Hashim; she is 55 years old, living in Basrah/Al-Qahera quarter, Moslem, married and having 5 children, and is a house-wife. Her next of kin is her daughter; Luma Mueen Nasir. The patient is admitted into the hospital on fourth of Maech/2012 chiefly complaining from left sided weakness for 30 minutes prior to her admission. Her history was started two days ago when the patient had noticed headache that was located to the back of her head, experienced on awakening of that day, dull-ache in nature, exacerbated by bending during praying and mild-moderate in severity; as the patient is hypertensive she used to get similar attacks of such headache in the previous three years. The headache persisted over that period and even gradually increased in severity till the patient had checked her blood pressure and discovered that it was so high two hours prior to admission. At that time she asked for medical advice and indented to visit the hospital; she however thought that her condition will improve in the next hours. Thirty minutes prior to her admission she noted difficulty in moving her left arm and leg with numb face, difficulty in speech, and spreading sensation of pins and needle; this weakness started suddenly, heaviness-like in nature, had no specific relieving or exacerbating factors, mild at the start then became more severe incapacitating her to move her left side unless with difficulty; she was able to move her right side and had not encountered feeling of rotation, swallowing difficulty, disturbance in vision, hearing, and smell, no movement abnormalities, no ocular abnormalities, and no vomiting. Immediately she had been rushed into the hospital were she received intravenous medication and underwent basic investigations; she felt with complete improvement 30 minutes after admission into the emergency unit; in the emergency unit her blood pressure was also too high.
Of note the patient had no prior similar attacks; had hypertension diagnosed on the basis of frequent high blood pressure measurement for the previous three years and kept on single daily oral tablet taken in the morning with very infrequent periodic check-up; she had two emergency unit admissions for high blood pressure and nose-bleeding before one year and six months after that. Peculiar to current complain, she started to use pain-reducing medications of her own accord to relieve her joint pains in the previous six months; she used them during the whole period of headache.

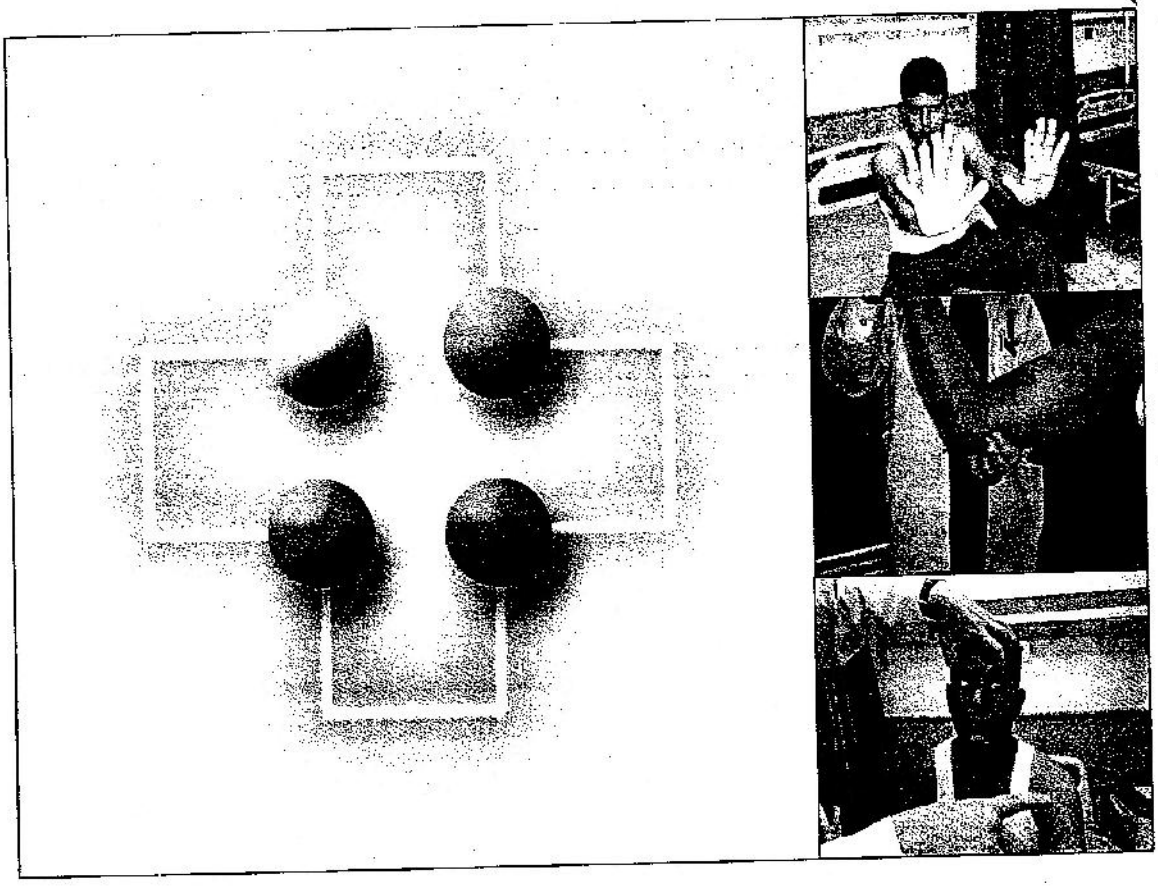
Regarding review of other systems:-
In the cardiovascular system, there was no dyspnoea, no PND, no orthopnoea, no platypnoea, no palpitation, no chest pain, no ankle edema or cyanosis.
In the respiratory system, there was no cough, no sputum, no hemoptysis, no hoarseness of voice and no wheezing.
In the GIT symptoms, no weight loss, no water brush, and no dry mouth. No nausea, regurgitation, or rumination. No heartburn or indigestion. No dysphagia or odynophagia, no hematemesis, melena or rectal bleeding, no abdominal pain or distension, no diarrhea or constipation, and no jaundice.
In the urological system, there was no frequency, urgency, or dysuria. No loin pain or hematuria, no hesitancy, terminal dribbling, retention, or incontinence. No change in the color, form, volume or odor of urine.
In the musculoskeletal system, no muscle stiffness, no joint pain, swelling, or limitation of joint movement, and no bone pain.
In the integumentary system, no change in the color of skin, skin rash, or skin itching. No nail changes, hair loss or hirsutism, and no oral/genital ulceration.
Regarding past medical history, the patient had hypertension; no prior history of diabetes mellitus, no ischemic heart disease, and no prior history of epilepsy or migraine.
Regarding past surgical history, she underwent no prior operation.
Regarding menstrual and gynecological history, she is Gravida 5, Para 5; she is postmenopausal 10 years ago.
Regarding drug history, she used single daily anti-hypertensive tablet and analgesics; no adverse effects for her anti-hypertensive medications had been encountered. No allergy to penicillin or sulfa-containing drug. She is not smoker, neither alcoholic nor addict.
Regarding socioeconomic history, the patient is living in urban area; purified water is the source of water; no indoor or out domestic animals.
Regarding family history, she is living with her husband and children in their own house; both her father and mother are dead due to ischemic heart disease.

Further reading

- Hutchison's clinical methods
- Davidson's principles and practice of medicine
- Harrison's principles of internal medicine
- Cecil principles of internal medicine
- Macleod's clinical examination
- Oxford Handbook of Clinical Examination – Practical Skills
- First Aid for the Internal Medicine Boards
- Clinical Examination - A Systematic Guide to Physical Diagnosis
- Textbook of Clinical Neurology
- Queen Square Textbook of Neurology
- Dictionary of Neurological Signs
- Clinical Neurology (McGraw-Hill, 2009)
- 2006 Adams and Victor's Principles of Neurology
- Baily and Love's short practice of surgery
- Gynecology by ten teachers
- Obstetrics by ten teachers
- Hamilton Baily's physical signs in clinical surgery
- Browse; signs and symptoms of surgical diseases
- Nelson's text book of pediatrics
- Bate's guide to physical diagnosis.
- Textbook of physical diagnosis.
- Oxford handbook of clinical examination/ practical skills.
- Kocher's Clinical Medicine for Students.

2

GENERAL EXAMINATION



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INTRODUCTION
The general examination is conducted under two headings; the general look to the patients and regional examination. These two steps should be presented simultaneously when a candidate is asked to generally examine the patient.

GENERAL LOOK TO THE PATIENT

It is essential to take permission from the patient before starting the examination saying for example:
Good morning (or how are you?), I m Dr. Ahmed, a third year medical student. Can I examine you?
Then ensure complete privacy to the patient by pulling the curtains around his/her bed. Undergraduate as well as postgraduate student should then practice the general look to the patient that should include Age group of the patient, Sex, Awareness, Decubitus of the patient in the bed, state of Comfortability, Body built, Dyspnea and tachypnea. These physical signs should always be presented at the start of the presentation. A useful mnemonic is A SAD CBD.

EXAMPLES

Young aged male patient, conscious, lying comfortable in dorsal decubitus position, of a thin body built; he is not dyspneic and not tachypneic.

Old aged female patient, lethargic, lying uncomfortable in lateral decubitus position, of a thin body built; she is not dyspneic and not tachypneic.

I. AGE OF THE PATIENT
In long case examination, the age can be inquired from the history while in short case examination; the candidate can determine the age group of the patient according to the appearance.

II. SEX OF THE PATIENTS
From the appearance of the patient one can determine the sex.

III. AWARENESS OR LEVEL OF CONSCIOUSNESS
Level of consciousness primarily reflects the patient's capacity for arousal or wakefulness. It is determined by the level of stimuli needed for the patient to be aroused. The patients may fall into one of the following states:

1	Alert	When you start the examination and greet the patient in a normal voice, an alert patient opens eyes, looks at you, and responds fully and appropriately to stimuli
2	Lethargic	When you start the examination and greet the patient in a normal voice, a lethargic patient is unresponsive. When you then greet the patient in a loud voice, a lethargic patient appears drowsy but opens eyes and looks at you, responds to questions, and then falls asleep
3	Obtunded	Shake the patient gently as if awakening a sleeper. An obtunded patient opens the eyes and looks at you, but responds slowly and is somewhat confused. The patient immediately reverts to his or her prior level of consciousness when the stimulation ceases
4	Stuporous	Only vigorous external stimuli can produce arousal e.g. squeezing the Achilles tendon, sternal pressure applied with a hard blunt object or supraorbital pressure from the examiner's thumb. These measures need of course permission from the examination committee
5	Comatose	Patients do not open their eyes utter sounds or obey commands in response to verbal or other stimuli

IV. DECUBITUS OF THE PATIENT IN THE BED
Decubitus (FIGURE 2 – 1) is the position of the patient in the bed. Patients may assume one of the following positions:

1	Dorsal decubitus	The patient lies on his/her back
2	lateral decubitus	The patient assumes either left lateral or right lateral position
3	Semi-recumbent posture	The head of the bed is elevated 45°. This position is adopted in patients with orthopnea
4	Prone position	The patient lies on his/her abdomen
5	Sitting position	The patient is sitting in the bed
6	Tripod position	The patient leans foreword and braces his/her hands or elbows on knees or a nearby table. This position stabilizes the shoulder girdle and helps the action of the accessory muscles in patients with severe dyspnoea.
7	Coiled-up decubitus	The patient lies on one side approximating his knees to his head. It is often noticed in patient with severe abdominal colic

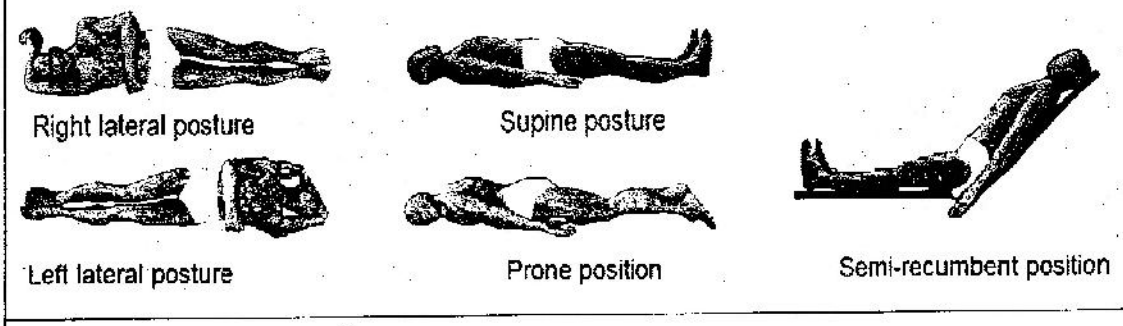


FIGURE 2 – 1. Some clinical positions

V. STATE OF COMFORTABILITY

The state of comfortability is presented as either well-looking and comfortable or ill-looking and uncomfortable.

VI. BODY BUILT (PHYSIQUE)

The patient may appear of a normal weight, obese, or under-weight (BOX 2 – 1). Obesity is developed when dietary intake had exceeded energy expenditure. Increased intra-abdominal fat causes central (abdominal, visceral, android or apple-shaped) obesity, which contrasts with subcutaneous fat accumulation causing generalized (gynoid, or pear-shaped); the former is more common in men (FIGURE 2 – 2). In adult, nutritional status is best assessed in term of BMI (Body Mass Index) which is derived from the formula Wt/Ht^2, measured in kg/m^2 (BOX 2 – 1). Obvious weight loss even when food intake has increased is seen in thyrotoxicosis, diabetes mellitus, lymphoma, and malabsorption. Wasting may be associated with chronic alcohol abuse, HIV infection, malignancy, and anorexia nervosa.

Height should be roughly equal to the fingertip to fingertip measurement of outstretched arms and twice the leg length from pubis to heel. Increase in height (Gigantism) may occur in patient with increased growth hormone secretion before puberty while a decrease in growth hormone results in dwarfism.

BOX 2 – 1. The relationship between body mass index and nutritional status

Condition	BMI
Underweight	<18
Normal	18.5 – 24.9
Overweight	25 – 29.9
Obese	30 – 39.9
Morbid obesity	≥ 40

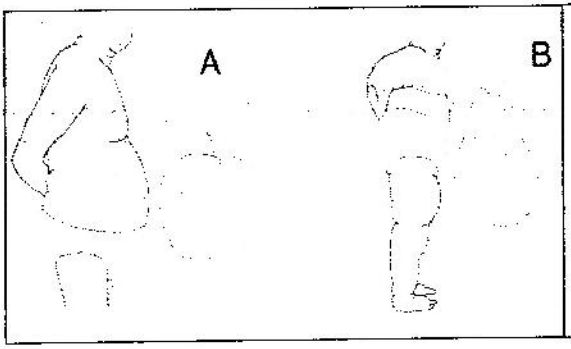


FIGURE 2–2. Types of obesity; in male (A) and in female (B).

VII. DYSPNOEA

Dyspnoea or breathlessness is an undue awareness of breathing. It is normal with strenuous physical exercise. It can be defined clinically as shortness of breath with some or all of the following:-

I. Abnormal increased inspiratory movement evident by:

- Dilation of alae nasi.
- Indrawing of suprasternal fossa, supraclavicular fossae, and intercostals spaces due to the use of accessory muscles (sternomastoids, platysma and pectoral muscles) of respiration to increase chest expansion during each inspiration. These muscles cause elevation of the shoulders with inspiration and aid respiration by increasing chest expansion. The use of accessory muscles is characteristic of patients with chronic obstructive pulmonary disease (COPD) and severe acute asthma.
- Abdominal paradox (Hoover's sign) that is indicative of diaphragmatic muscle fatigue.
- Tracheal descent during inspiration (tracheal tug).

II. Abnormal increased expiratory movement evident by:

- The patients assume upright or sitting position grasping a bed table or the back of the chair (Tripod position). This position stabilizes the shoulder girdles and helps the action of the accessory muscles.
- The patients exhale through their mouth with pursed lip and this is called (pursed lip breathing). Pursed-lip breathing provides a positive intra-airway pressure that is thought to decrease airway closure and air trapping and to aid gas exchange. Patients often discover this maneuver on their own or learn it through formal instruction by healthcare providers, although the exact benefit of this technique has not been established.

Tachypnea is defined as an increase in respiratory rate. When there is dyspnoea, there is tachypnea.

REGIONAL EXAMINATION OF THE PATIENT

The regional approach will help the clinician not to miss any part of the body as well as it simplifies the sequence of examination. The approach is variable but most of the current medical book agreed to start the examination firstly of the hands and as provided below. We used to present any case taking particular consideration to Jaundice, Anemia, Cyanosis, Clubbing, Oedema, and Lymphadenopathy. These signs are collectively known as JACCOL.

THE HANDS

The hands are simply examined for dorsal abnormalities, palmar abnormalities, and for tremor. Names of the fingers are shown in FIGURE 2-3. POSTURE may give a clue to the underlying disease. Flexed hand and arm occurs in hemiplegics; claw hand in ulnar nerve palsy (FIGURE 2 – 4); wrist drop in radial nerve palsy (FIGURE 2 – 4); and finger drop in patients with posterior interosseous nerve palsy. The hands in carpopedal spasm (tetany) adopt a characteristic position (FIGURE 2 – 4). The metacarpophalangeal joints are flexed, the interphalangeal joints of the fingers and thumb are extended, and there is opposition of the thumb. This occurs due to hypocalcemia. Rheumatoid arthritis most often causes symmetric arthritis with characteristic involvement of certain joints such as the proximal interphalangeal and metacarpophalangeal joints. The distal interphalangeal joints are rarely involved. Synovitis of the wrist joints is a nearly uniform feature of rheumatoid arthritis and may lead to limitation of motion, deformity, and median nerve entrapment (carpal tunnel syndrome). With persistent inflammation, a variety of characteristic joint changes develop (BOX 2 – 2). Some of these joint changes are shown in FIGURE 2 – 5.

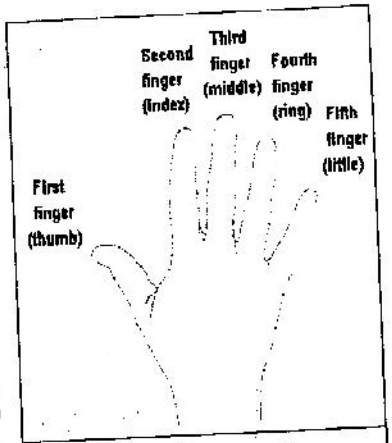
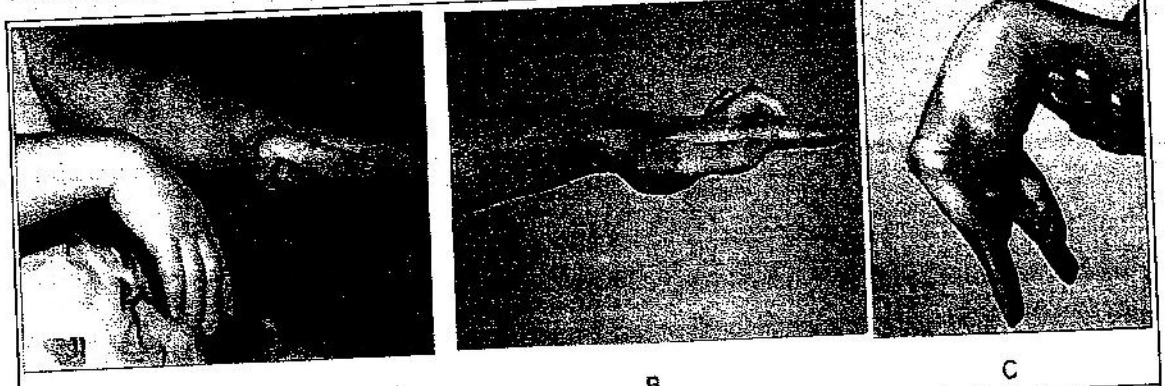


FIGURE 2 – 3. Names of the fingers.



A B C

FIGURE 2 – 4. Characteristic hand signs in radial nerve palsy (A), ulnar nerve palsy (B), and carpal spasm (C)

BOX 2 – 2. Typical rheumatoid arthritis hand signs

1	Symmetrical swelling of the metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints
2	Sparing of the distal interphalangeal (DIP) joints.
3	Swan-neck deformity means hyperextension of the PIP joints, with compensatory flexion of the DIP joints
4	Boutonnière deformity means flexion contracture of the PIP joints and extension of the DIP joints
5	"Z" deformity of the thumb due to palmar subluxation of the proximal phalanx
6	Radial deviation at the wrist with ulnar deviation of the digits
7	Wasting of the small muscles of the hands
8	Dorsal subluxation of the ulnar styloid process of the wrist
9	Trigger fingers
10	Palmar erythema and palmar creases pallor
11	Splinter hemorrhage
12	Rheumatoid nodules

CLINICAL CLUE

Manifestations of hypocalcemia are primarily related to increased neuromuscular irritability. Tetany is the classic sign of hypocalcemia, but it is not always present. Paresthesias are more common and often first occur around the mouth or in the fingertips. They may progress to overt muscle spasm in the face and extremities, the latter typified by carpopedal spasm. The term latent tetany refers to signs elicited by provocative stimuli such as ischemia (Trousseau's test: induction of carpal spasm within 3 minutes of inflating a sphygmomanometer above systolic blood pressure) or percussion of the facial nerve, about 3 – 5 cm below and in front the ear, to induce involuntary contraction of the facial muscles including the corner of the mouth, nose, and eye (Chvostek's sign).

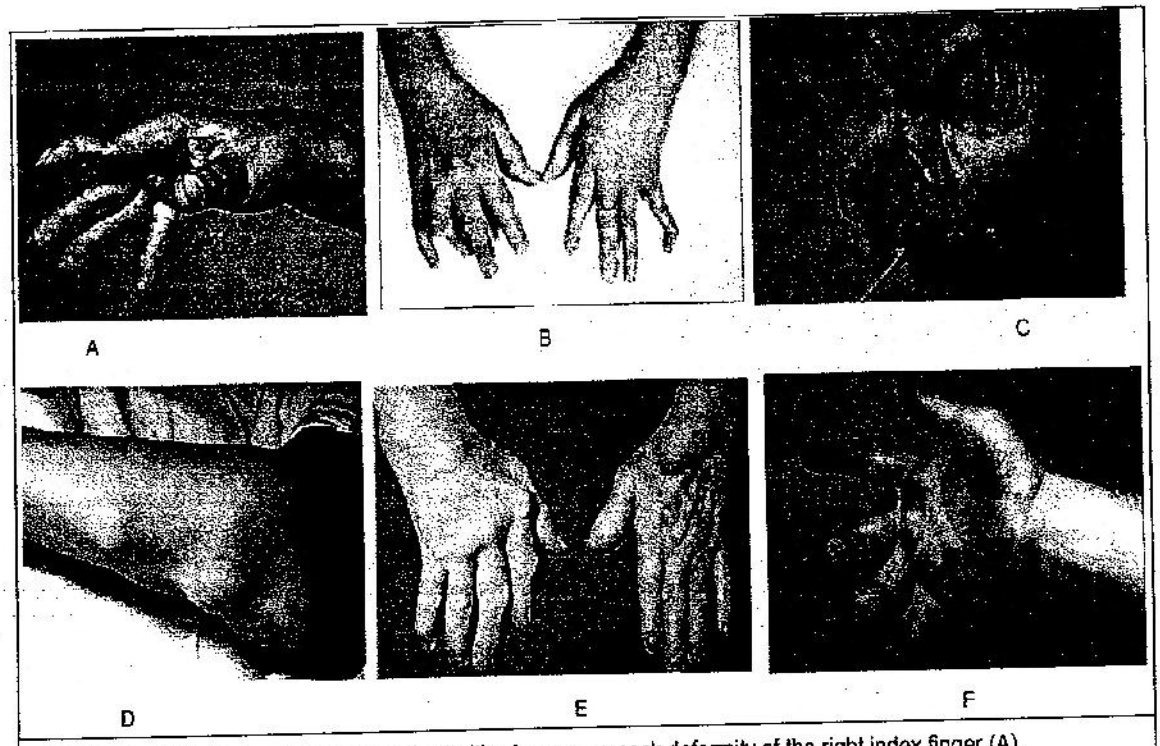


FIGURE 2 – 5. Patients with rheumatoid arthritis show swan neck deformity of the right index finger (A), boutonnière deformity or button hole deformity of the right ring finger (B), Z – shape deformity of the right thumb (C), rheumatoid nodules (D), ulnar deviation of the right fingers (E), and palmar erythema (F).

Typical features of osteoarthritis (FIGURE 2 – 6) in the hands include Bouchard's nodule at the proximal interphalangeal (IPJs) joints, Heberden's nodule at the distal IPJs, and thumb base squaring. Dupuytren contracture (FIGURE 2 – 6) is a palmar thickening associated with flexion contracture of the ring finger and the litile finger. This deformity is associated with chronic alcoholic misuse, diabetes mellitus, familial cases, chronic vibration injury, and drugs (phenytoin and phenobarbital).

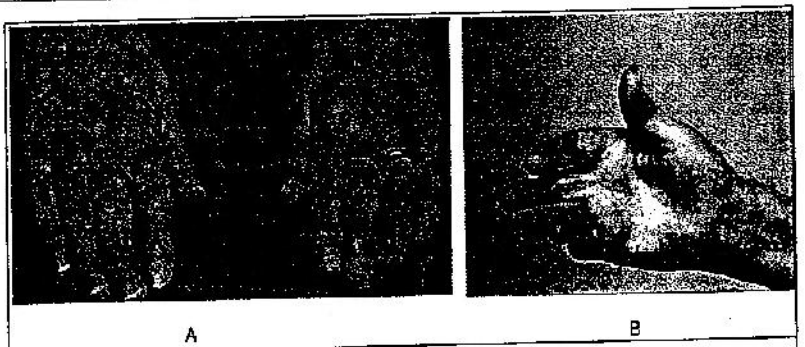
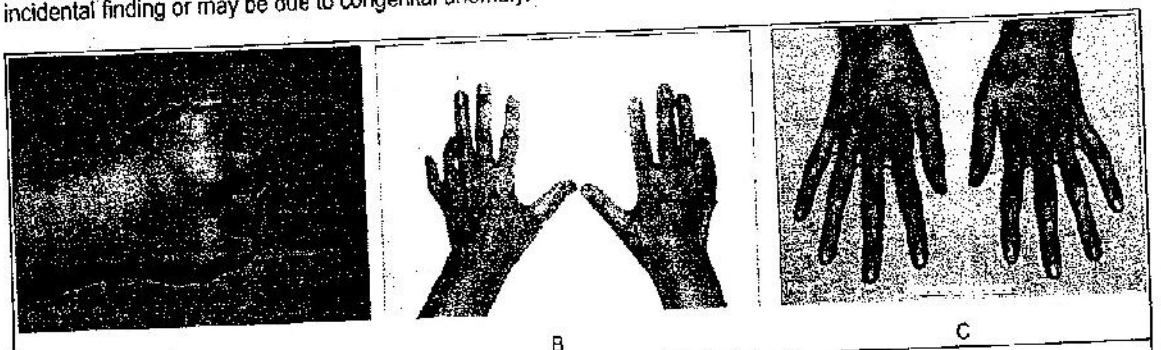


FIGURE 2 – 6. Heberden's nodes are seen on the distal interphalangeal joints and Bouchard's nodes are seen on the proximal interphalangeal joints (A) and typical Dupuytren contracture (B).

SHAPE of the hand is also valuable. Trauma is the most common cause of hand deformity; long thin fingers are seen in arachnodactyly characteristic of Marfan's syndrome. Patients with Marfan's syndrome may be recognized by their tall stature, dislocated lenses and a high-arched palate; nevertheless, hands with arachnodactyly (FIGURE 2 – 7) provide convincing supportive evidence. The syndrome of pseudohypoparathyroidism (tissue unresponsiveness to

parathyroid hormone) may be suspected from the facial features (e.g. mental retardation, short stature, short neck, round face and abnormal teeth) but the hands can offer reassuring clues. The third, fourth and fifth metacarpals, or any one or two of them, are short, which can be revealed by asking the patient to make a fist (FIGURE 2 – 7). In the Lawrence-Moon-Biedl syndrome, the presence of polydactyly (extradactyly or extra digit) (FIGURE 2 – 7) is associated with obesity, mental retardation and hypogonadism. It is important to look for these additional features because polydactyly can be present in a normal subject as an incidental finding. Syndactyly (fused digit) can be an incidental finding or may be due to congenital anomaly.



A B C

FIGURE 2 – 7. Patients with pseudohypoparathyroidism when asked to make a fist a dimple will appear owing to short metacarpophalangeal joint (short fourth metacarpophalangeal joint) as in A, polydactyly may be an incidental finding (B), and arachnodactyly (C) is associated with Marfan's syndrome.

CLINICAL CLUE

Differential diagnoses of rheumatoid – like hands are:

1. Rheumatoid arthritis.
2. Systemic lupus erythematosus. The deformities here are reducible due to affection of the cartilages rather than the joints and called 'Jaccoud's arthropathy'. Reduction is performed by pulling the distal end of the fingers.
3. Osteoarthritis. There are Heberden's nodules and Bouchard's nodules.
4. Psoriatic arthropathy. There may be skin and nail changes consistent with psoriasis.

COLOUR of the hand should also be noted. A brownish stain on the fingers and nails in cigarette smokers is caused by tar, not nicotine (FIGURE 2 – 8); bluish discoloration of the ends of the fingers indicates cyanosis (FIGURE 2 – 8); white color in digits suggests Raynaud's phenomenon (FIGURE 2 – 8).

A B C

FIGURE 2 – 8. Some clinical findings including patients with Raynaud's phenomenon showing the pale phase (A), tar staining of the fingers (B), and cyanosed hands (C).

Raynaud's phenomenon is a sequence of changes usually seen in the hands where the digital arterioles get spasm in response to cold or emotional stimuli so that the fingers become pale, then cyanosed but when spasm passes off, the fingers became hyperemic and red (a useful mnemonic is PCR). The pallor represents the ischemic phase of the

phenomenon and results from vasospasm of digital arteries. The cyanosis results from the deoxygenated blood present in the vessels. A sensation of cold or numbness or paresthesia of the digits often accompanies the phases of pallor and cyanosis. With rewarming, the digital vasospasm resolves, and blood flow into the dilated arterioles and capillaries increases dramatically. This "reactive hyperemia" imparts a bright red color to the digits and patients often experience a throbbing, painful sensation during the hyperemic phase. Although the triphasic color response is typical of Raynaud's phenomenon, some patients may develop only pallor and cyanosis; others may experience only cyanosis. Raynaud's phenomenon is classified into primary or idiopathic Raynaud's phenomenon (Raynaud's disease) and secondary Raynaud's phenomenon (Raynaud's syndrome). The differentiation is presented in BOX 2 – 3.

BOX 2 – 3: The differentiation between Raynaud's disease and Raynaud's syndrome

	Characteristics	Primary	Secondary
1	Associated diseases	No	Yes
2	Age at onset	Younger (<30 yr)	Older (>30 yr)
3	Sex	Female	Male
4	Symmetry	Symmetrical	Asymmetrical
5	Family history	Yes	No
6	Nailfold capillaries	Normal	Large, tortuous with "dropout"
7	Autoantibodies	Negative or low titers	Frequent
8	Digital gangrene	Rare; only superficial	Common

Common causes of secondary Raynaud's phenomenon (Raynaud's syndrome) are (mnemonic: COLD HAND):
Cryoglobulinemia
Obstruction (thoracic outlet syndrome)
Lupus and other connective tissue disease (scleroderma, dermatomyositis, polymyositis, and Sjögren's syndrome)
Diabetes mellitus
Hematologic problems (polycythemia, leukemia, etc)
Arterial problems (atherosclerosis and thromboangiitis obliterans)
Neurologic problems (Intervertebral disk disease, syringomyelia, stroke, carpal tunnel syndrome)
Drugs (Ergot derivatives, methysergide, β-adrenergic receptor blockers, bleomycin, cisplatin)

SIZE of the hands should also be noted as for example in patient with acromegaly, there is overgrowth of the facial skeleton, coarsening of features and an increase in soft tissue size (FIGURE 2 – 9).

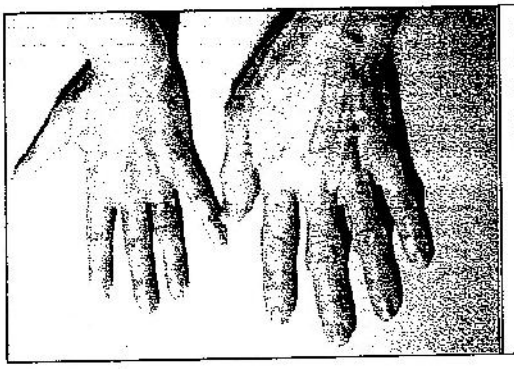


FIGURE 2 – 9. Normal (left) and acromegalic hand (right).

TEMPERATURE is assessed roughly by touching the dorsal surface of the patient's hands with the dorsal surface of your fingers (Rule: back over back). Warm hand occurs in peripheral vasodilatation due to fever, acute exacerbation of COPD, and in thyrotoxicosis. Cold hands may be noticed due to peripheral vasoconstriction e.g. ischemia, hypotension, and vasospasm (Raynaud's phenomenon).

THE NAILS of both hands should be checked. The nail plate arises from the nail matrix and lies on the nail bed. The keratinous plate is produced by cells of the matrix and to much lesser extent the bed (FIGURE 2 – 10). Finger nails grow about 1 cm every 3 months and toes nails grow at about one-third of this rate.

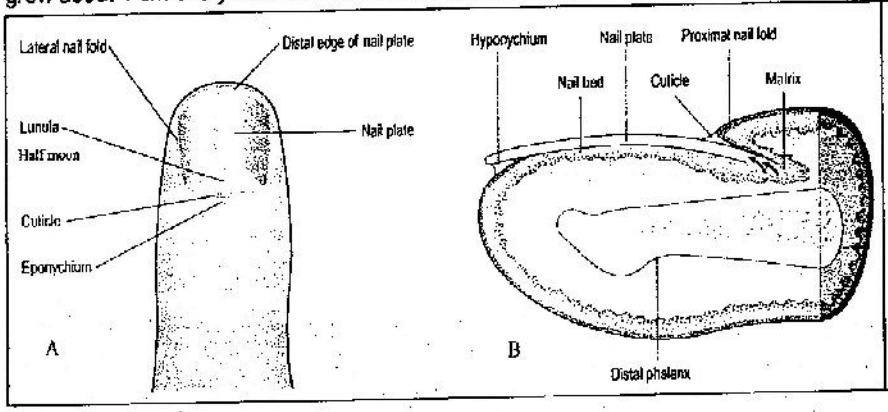


FIGURE 2 – 10. Structure of the nail in dorsal view (A) and lateral view (B).

In clinical practice the disorders of the nail (FIGURE 2 – 11 and 2 – 12) may take one or more of the following:

1	Nail biting	It is thought to be a manifestation of an anxious personality.
2	Finger clubbing	Discussed below
3	Koilonychia (Lindsay sign)	Refers to soft thin, easily broken (brittle) and spoon–shaped nail that may occur in iron deficiency anemia, lichen planus, hemochromatosis, and repeated detergent exposure. It may however be idiopathic or congenital. Koilonychia is divided into two stages: Stage I: there is flattening of the nail plates. Stage II: the edges became everted upward and the nail appears concave giving to the term spoon shaped nails. Some clinicians use the water-drop test to confirm such an abnormality.
4	Beau's lines	Transverse grooves which appear at the same time on all nails a few weeks after an acute illness due to temporary arrest of nail growth.
5	Paronychia	Means inflammation of the nail folds and appears as tender red swollen spots that may give rise to pus accumulation
6	Leukonychia	Whitening of the nail that presents as total whitening due to hypoalbuminemia or as white lines. These lines are either Muehrck's lines that disappear with pressure to the nail (due to chronic hypoalbuminemia) or Mees' lines that do not disappear with pressure (due to Arsenic exposure). Trauma stills the most common cause of white lines.
7	Finger pitting	Seen most commonly in Psoriasis, alopecia areata, eczema and fungal infection.
8	Splinter hemorrhages	Linear hemorrhages in the nail occur due to trauma (most common cause), vasculitis, rheumatoid arthritis, infective endocarditis, psoriasis, and trichinosis. True splinter hemorrhages are three or more in number and located 3 – 5 mm from the free edge.
9	Onycholysis	Separation of nail plate from nail bed and occur in trauma, psoriasis, and thyroid diseases.
10	Yellow nail	Seen in yellow nail syndrome which is a triad of slowly growing yellow nails, pleural effusion, and bronchiectasis.
11	Red half moons	Red lunulae due to congestive heart failure.
12	Blue half moons	Bluish discoloration of the normally white lunula suggests chronic cyanotic state e.g. Eisenmenger's syndrome. Darker blue lunulae are sometimes found in Wilson's disease, hemochromatosis, and in patients on antimalarial drugs or minocycline.
13	Half and half nails	White proximally and red-brown distally seen in uremia and normally due to aging.

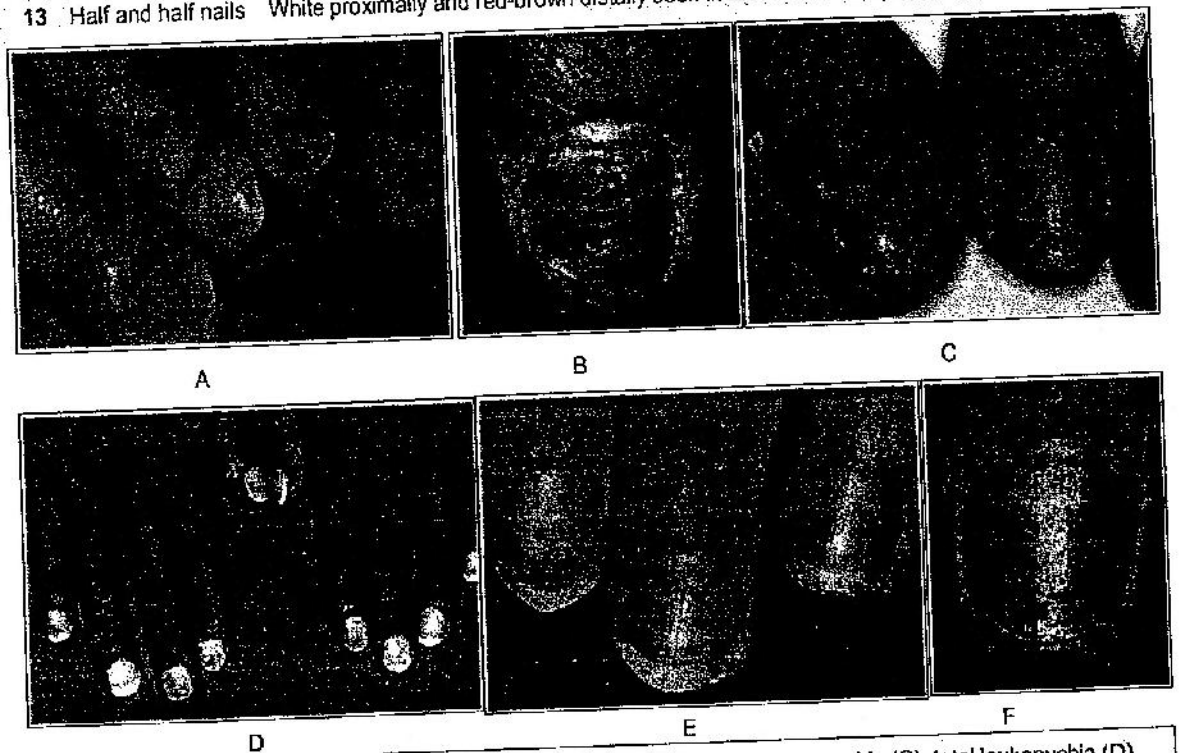


FIGURE 2 – 11. Various nail disorders as Koilonychia (A), Beau's lines (B), Paronychia (C), total leukonychia (D), Muehrck's lines (E), and nail pitting (F).

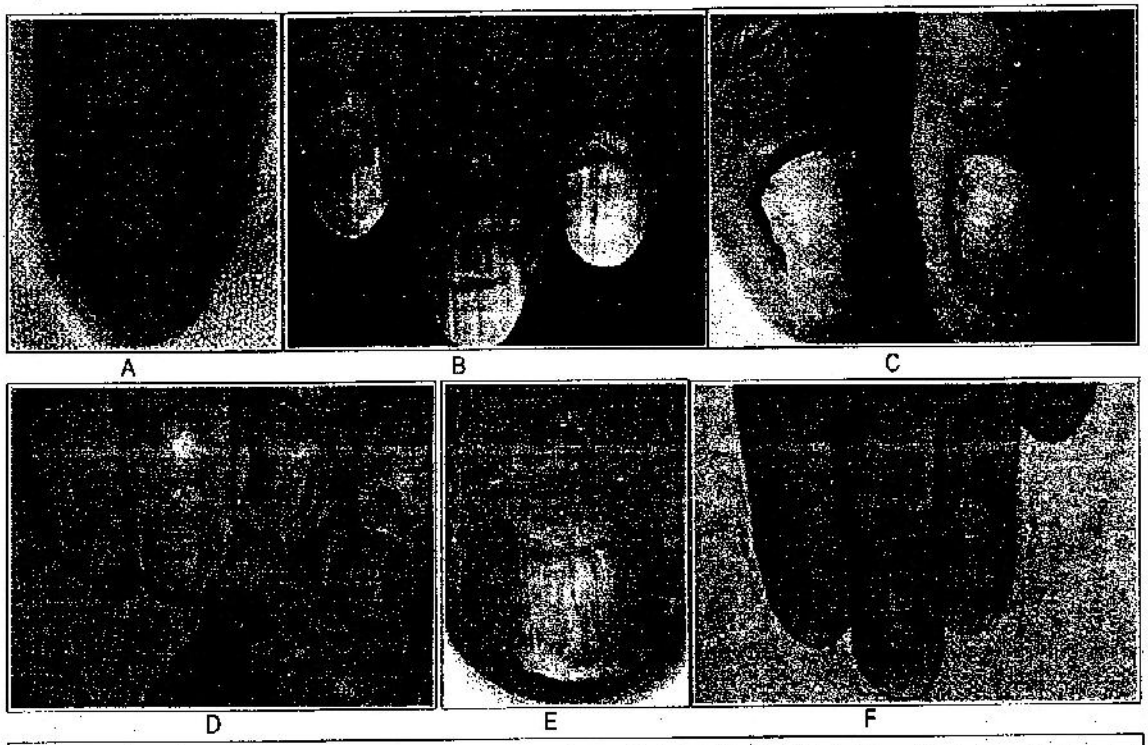


FIGURE 2 – 12. Various nail disorders including splinter hemorrhages (A), onycholysis (B), yellow nails (C), blue half moon (D), red half moon (E), and half and half nail (F).

Digital clubbing is defined as increased curvature of the nails with selective bullous enlargement of the distal segments of the fingers (and toes) due to proliferation of connective tissue particularly on the dorsal surface. There is also increased sponginess of the soft tissue at the base of the nail. Clinical stages of finger clubbing are:

1. Stage I In this stage the angle between the nail bed and adjacent skin of the finger (nail bed angle) is obliterated. Schamroth's window test involves placing of both finger nails in apposition (FIGURE 2 – 13): in normal individuals a distinct aperture or window, usually diamond-shaped, is formed at the base of the nail bed; clubbing obliterates this window (FIGURE 2 – 13). A critical comparison of clubbed finger with the profile of a normal finger will show that the angle between the proximal nailfold and the curved nail plate (Lovibond's 'profile sign') is increased from the normal 160° to over 180° (FIGURE 2 – 14). In the normal finger, the distal phalanx forms an almost straight line with the middle phalanx; whereas in the clubbed finger this angle is reduced to around 160° (Curth's modified 'profile sign') (FIGURE 2 – 14).
   A simple method to detect clubbing is the measurement of the phalangeal depth ratio (FIGURE 2 – 15). In normal finger the distal phalangeal depth is smaller than the interphalangeal depth. In clubbing this relationship is reversed and becomes more than one.

STEPS OF EXAMINATION

Perform Schamroth's tests to the thumbs

View the index or middle fingers from the radial side (FIGURE 2 – 16) to look at the Lovibond's 'profile sign' and Curth's modified 'profile sign'. Measure then the phalangeal depth ratio

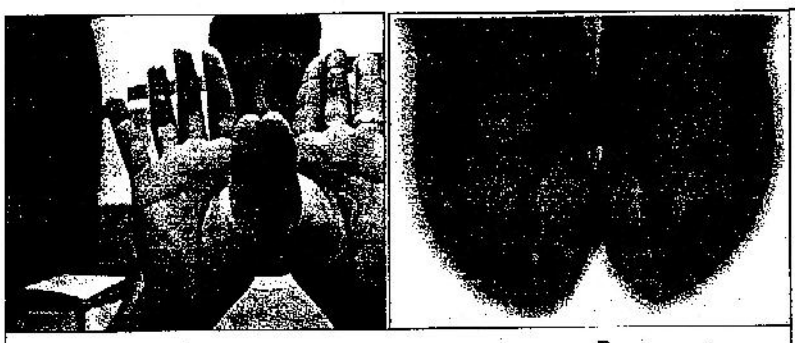


FIGURE 2 – 13. Clinical application of Schamroth's test (A) and an obliteration of the 'window' (B).

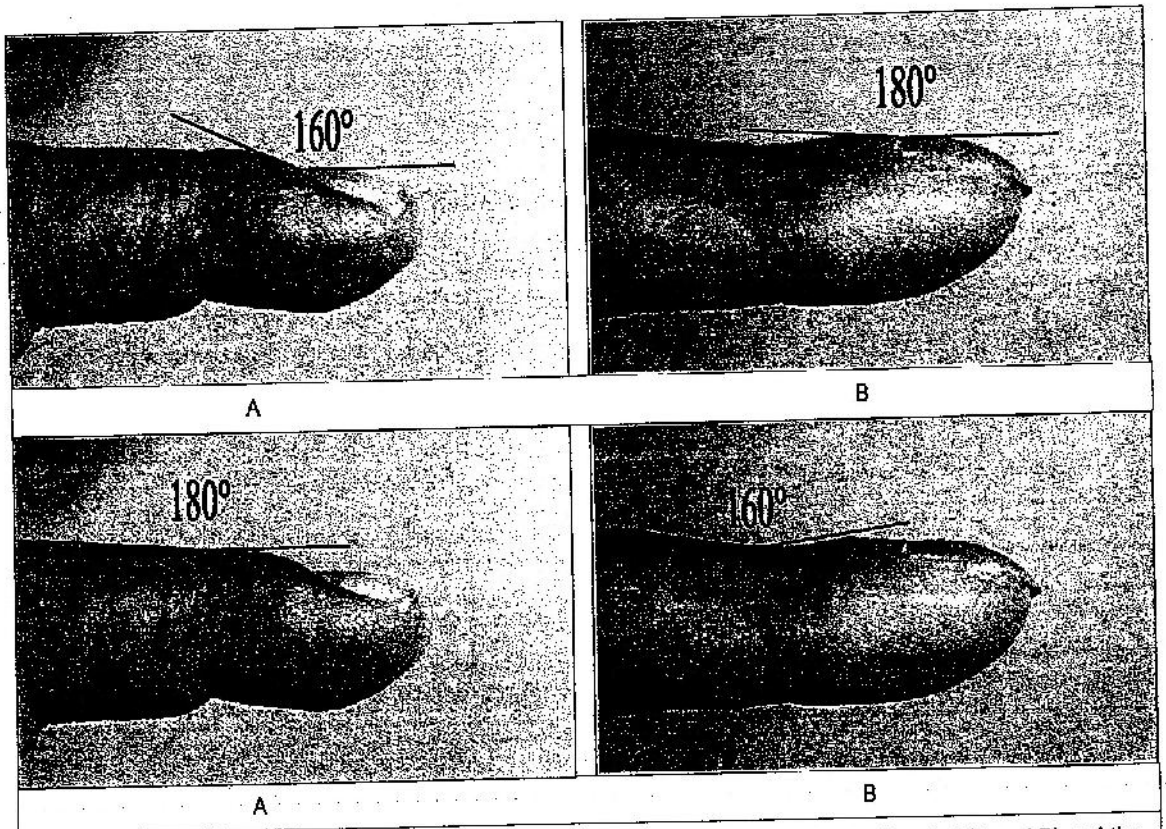


FIGURE 2 – 14. Viewing the fingers from the radial side to determine the Lovibond's 'profile sign' (A and B) and the Curth's modified 'profile sign' (C and D).

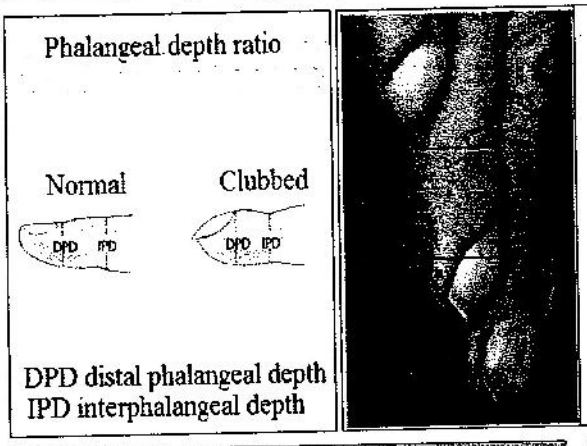


FIGURE 2 – 15. Measurement of the phalangeal depth ratio in both normal and clubbed fingers. In the latter the ratio is more than one (a > b).

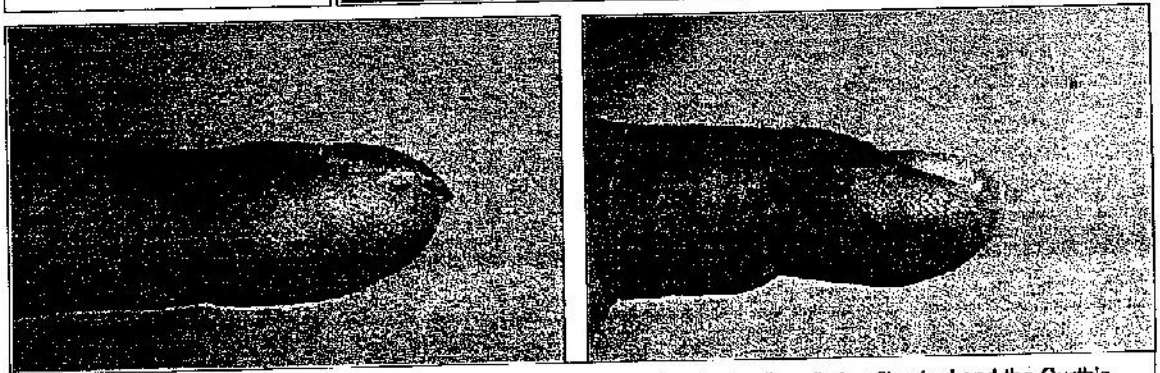
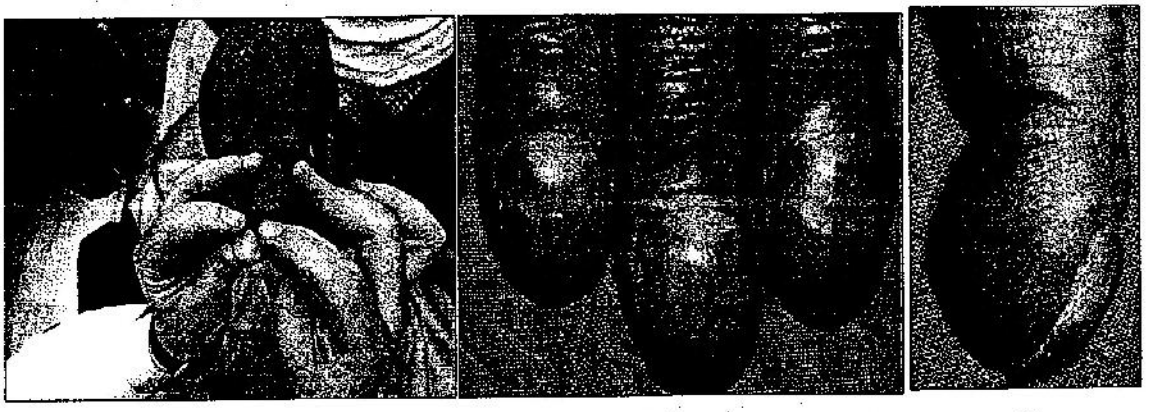


FIGURE 2 – 16. Viewing the fingers from the radial side to determine the Lovibond's 'profile sign' and the Curth's modified 'profile sign'.

2 Stage II In this stage the nail loses its longitudinal ridges and becomes convex from above down ward as well as from side to side.

3 Stage III In this stage there is a fluctuation of the nail bed. To elicit fluctuation, place the patient's finger on the pulp of your thumb and hold in this position by gentle pressure with the tip of your middle fingers over the proximal phalangeal joint. Then palpate the finger over the base of the nail with the tips of your two index fingers (FIGURE 2 – 17). A spongy feel confirm fluctuation.

4 Stage IV In this stage the terminal segment of the fingor becomes like the end of a drum stick (FIGURE 2 – 17).



A B C

FIGURE 2 – 17. The fluctuation test (A) and two views (B and C) of finger clubbing.

Clubbing may be hereditary, idiopathic, or acquired (BOX 2 – 4). Sometimes it is not uncommon to notice a unilateral clubbing. A unilateral clubbing may occur in hemiplegics and infected arteriovenous shunt.

BOX 2 – 4. Causes of digital clubbing

Respiratory	Alimentary
Primary and metastatic carcinoma Intrathoracic suppuration • Bronchiectasis • Empyema • Lung abscess Idiopathic pulmonary fibrosis (cryptogenic fibrosing alveolitis) Asbestosis	Hepatic cirrhosis Ulcerative colitis Crohn's disease Coeliac disease
Cardiovascular	**Other**
Cyanotic congenital heart disease Bacterial endocarditis	Familial clubbing Occupational e.g. jackhammer operators

In patients with patent ductus arteriosus complicated by Eisenmenger syndrome, the severe pulmonary vascular disease results in reversal of the blood flow through the ductus; unoxygenated blood is shunted to the descending aorta; and the toes, but not the fingers, become cyanotic and then clubbed, a finding termed differential clubbing.

CLINICAL CLUE

Differential diagnoses of digital clubbing (FIGURE 2 – 18) are:

5. Hypertrophic pulmonary osteoarthropathy (HPOA) is rare condition and almost always associated with lung cancer, usually squamous cancer. Pronounced clubbing of fingers and toes occurs, with pain and swelling affecting the wrists and ankles. X-rays of the distal forearm and lower legs show subperiosteal new bone formation separate from the cortex of the long bones
6. Thyroid acropachy which refers to a state of digital condition Indistinguishable form clubbing found in <1% of patients with Graves' disease. It is so strongly associated with thyroid dermopathy that an alternative cause of clubbing should be sought in a Graves' patient without coincident skin and orbital involvement.

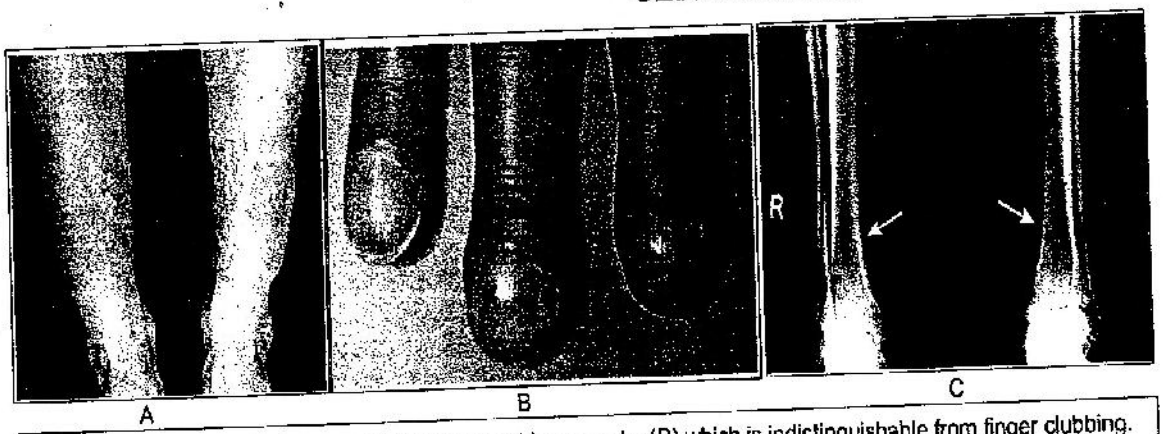


FIGURE 2 – 18. Thyroid dermopathy (A) and thyroid acropachy (B) which is indistinguishable from finger clubbing. In patient with hypertrophic pulmonary osteoarthropathy there is new periosteal bone formation (C).

The PALM of the hands should then be examined. Palmar creases are pale in anemia; if the palmar creases are lighter in color than the surrounding skin when the hand is hyperextended, the hemoglobin level is usually < 70 g/L (7 g/dL). Palmar erythema (FIGURE 2 – 5; F and 2 – 19) is a characteristic bright red eminences and pulps of the finger that occur in pregnancy, thyrotoxicosis, hepatic failure, or may be idiopathic. Palmar thickening occurs in eczema, familial Tylosis syndrome (FIGURE 2 – 19), dermatomyositis (called mechanic's hands), Keratoderma blennorrhagicum, and in Naxos disease.

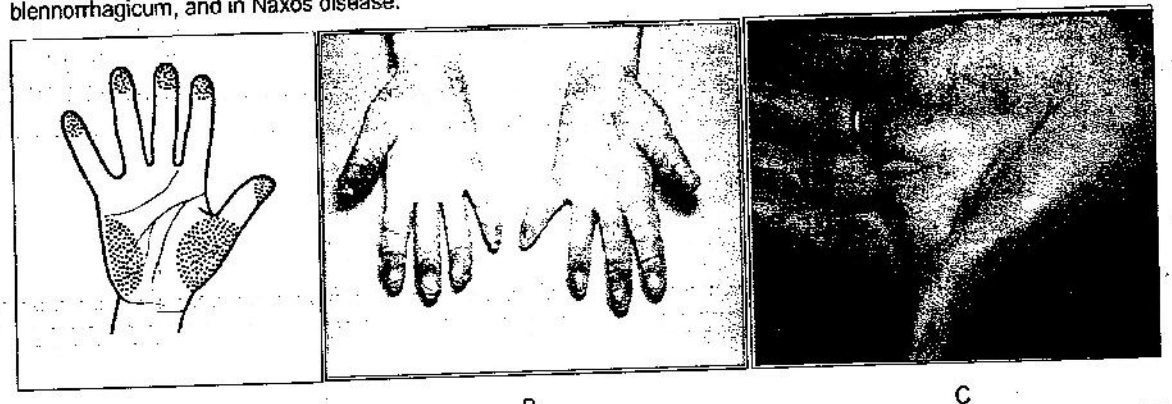
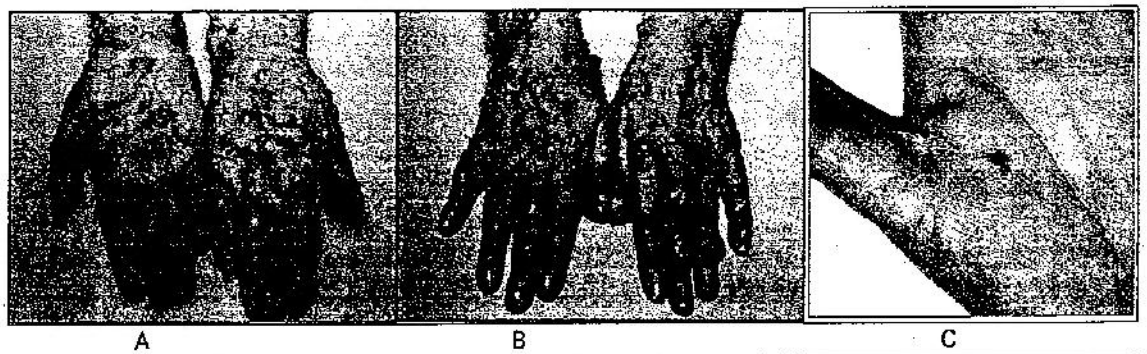


FIGURE 2 – 19. An illustration for the sites discolored red in palmar erythema (A). Keratotic palms in patients with Tylosis (B). Palmar creases may be pigmented in patient with Addison's disease (C).

CLINICAL CLUE

Familial Tylosis syndrome is characterized by thick skin with painful fissuring and is associated with squamous carcinoma of the esophagus. In dermatomyositis, the lateral and palmar areas of the fingers may become rough and cracked, with irregular, "dirty" horizontal lines, resembling mechanic's hands. Keratoderma blennorrhagicum occurs in patients with reactive arthritis and consists of vesicles that become hyperkeratotic and ultimately forming a crust before disappearing. They are most common on the palms and soles but may occur elsewhere as well. Naxos disease is a triad of arrhythmogenic right ventricular dysplasia, palmar/plantar keratosis and woolly hair. It is associated with sudden cardiac death in adolescents and young adults.

Palmar pigmentation may suggest Addison's disease (FIGURE 2 – 19); yellow palm occurs in carotenoderma and jaundice. In patients with neurofibromatosis (von Recklinghausen's disease), pedunculated or sessile tumours of varying size may occur on the hands or elsewhere (FIGURE 2 – 20). The hands may show numerous telangiectasias in Osler-Weber-Rendu syndrome (hereditary hemorrhagic telangiectasia. Osler's nodes are painful erythematous lumps in the pulps of the fingers and toes, palms of the hands and soles of the feet. This sign occurs in infective endocarditis. Janeway lesions (FIGURE 2 – 20) are non-tender erythematous maculopapular lesions contalning bacteria, which occur on the palms or pulps of the fingers in patients with infective endocarditis.

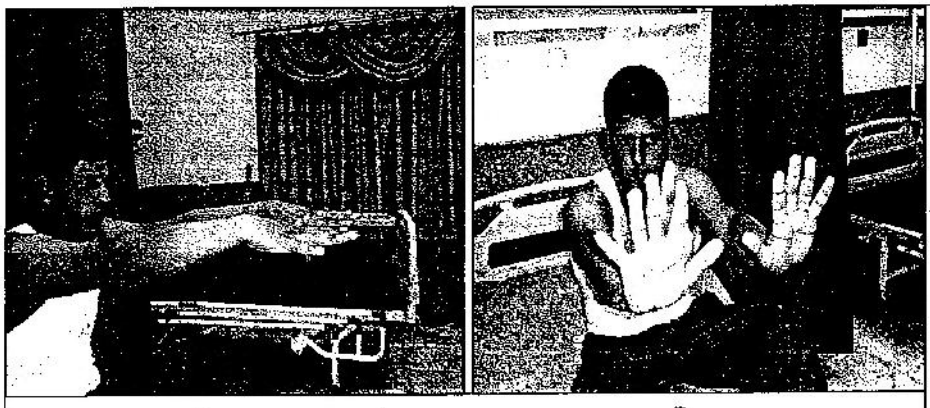


A B C

FIGURE 2 – 20. In patients with neurofibromatosis there are numerous (A and B) tumors of varying size. Non-tender erythematous maculopapular lesions in patient with infective endocarditis (C) called Janeway lesion.

Excessive SWEATING of the hands may be idiopathic or psychogenic in origin or can be found in thyrotoxicosis. Finally check the patient for the presence of HAND TREMOR. Tremor is defined as rhythmical contraction and relaxation of muscle group. Two types of hand tremor should the candidate looked (FIGURE 2 – 21) for:

1 Postural tremor	It is clinically separated into fine or coarse tremor.
Examination technique	Ask the patient to place hands straight out in front and spread the finger.
Causes	Anxiety Drugs e.g. ß-agonists (salbutamol), theophylline, and tricyclic antidepressants Excessive tea or coffee drinking Endocrine e.g. thyrotoxicosis, pheochromocytoma, and hypoglycemia
2 Flapping tremor (asterixis)	It is defined as a repetitive partial flexion of the wrist on wrist extension
Examination technique	Ask the patient to hold out the arms with the hands extended at the wrists. Look for a jerky, flapping tremor
Alternatively	Ask the patient to squeeze your index and middle fingers and maintain this for 30-60 seconds. Patients with a flapping tremor cannot maintain the posture.
Causes	Organ failure e.g. respiratory, cardiac, or renal Metabolic disturbances e.g. hypoglycemia, hypokalemia, or hypomagnesemia Drugs e.g. antiepileptic (barbiturates, sodium valproate, phenytoin, or carbamazepine), and metoclopramide CNS e.g. intracerebral hemorrhage, subdural hematoma, subarachnoid hemorrhage, or cerebral ischemia



A B

FIGURE 2 – 21. The clinical technique to perform postural tremor (A) and flapping tremor (B) (asterixis).

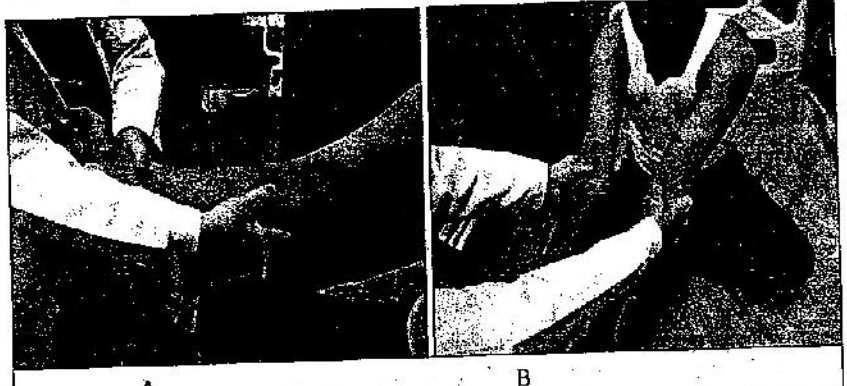
After hand examination the candidate should examine both arms looking in particular for radial and brachial pulses abnormalities, epitrochlear lymphadenopathy, sites of insulin and heparin injection, and finally for axillary lymphadenopathy. Since most of our Moslem women are covering their arms with clothes especially in winter season the candidate should first ask the patient about cannula placed under the clothes and then to take a permission from the patient that you are going to take off the arm clothes up to the upper part of the arms.

THE RADIAL PULSE

The details of radial pulse examination are discussed under the heading vital signs.

THE BRACHIAL PULSE

The brachial pulse is felt with the thumb of the right hand compressed against humerus just above the antecubital fossa medial to biceps tendon (FIGURE 2 – 22). Brachial pulse is used to detect the volume of blood. When searching for a very weak pulse as in the presence of peripheral vascular disease, it is better to use the fingers rather than thumb. Compare after that the right brachial pulse with the left.



A B

FIGURE 2 – 22. The technique of brachial artery detection (A) and simultaneous comparison (B) of both brachial arteries.

EPITROCHLEAR LYMPH NODES

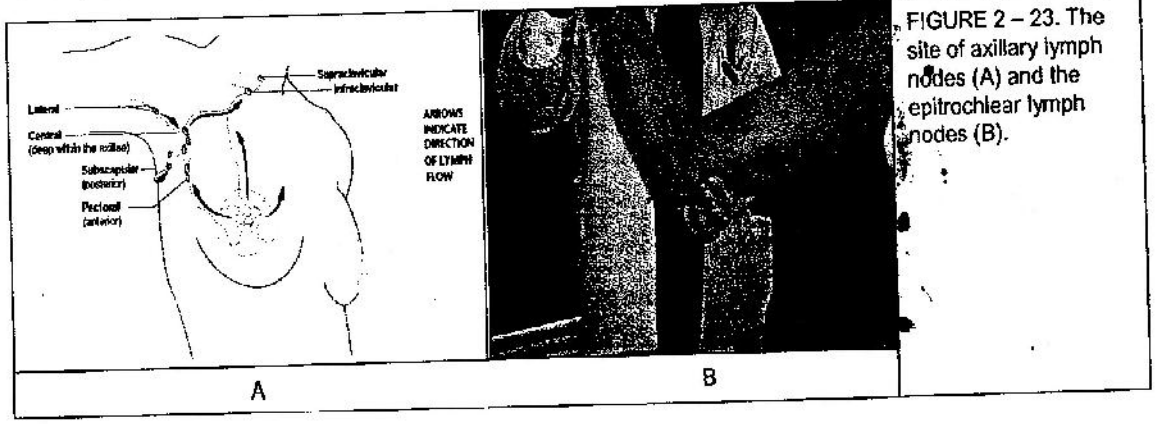
First flex the right arm of the patient to a right angle in order to relax surrounding structures and then palpate the gland (FIGURE 2 – 23) in the groove above and posterior to the medial epicondyle of the humerus. Bilateral enlargement of this gland suggests a generalized lymphadenopathy while unilateral enlargement suggests infection of the hand, wrist and forearm.

AXILLARY LYMPH NODES

Of the axillary lymph nodes, the central nodes are palpable most frequently. The examination of the axillary lymph nodes is usually conducted through the following steps:

1 Take the patient's right elbow with your right hand and examine using your left hand for the right side axillary's lymph node (Rule: right – right) and vice versa for the left side.

2 Palpate the anterior group firstly then posterior group then lateral group and then central group (FIGURE 2 – 19)



A B

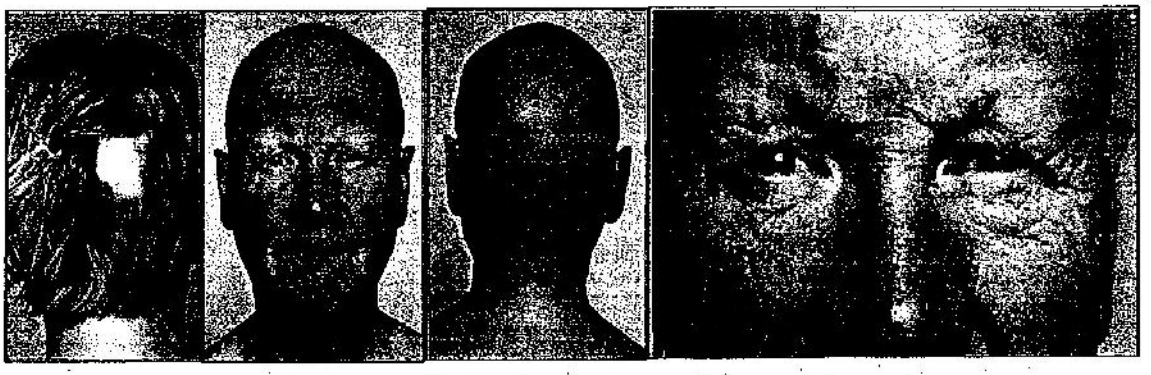
FIGURE 2 – 23. The site of axillary lymph nodes (A) and the epitrochlear lymph nodes (B).

CLINICAL CLUE

The presentation and causes of lymphadenopathy are described together with lymph nodes of the head and neck.

THE SCALP

Each hair follicle passes through regular cycles of growth phase (anagen) and resting/shedding phase (telogen). Anagen in the scalp lasts up to 5 years while telogen is mush shorter lasting about 3 months. Normally 85% of scalp hairs are in anagen and 15% in telogen. The term (catogen) is the conversion stage from active to resting stage and lasts few days. On the scalp there are about 100000 hairs; normal daily shedding is about 100 hairs. Temporal recession is common in male but in female it may suggest virilization. ALOPECIA is a term used to describe hair loss. Alopecia areata describes patchy and localized (FIGURE 2 – 24) hair loss; alopecia totalis describes total scalp baldness; and alopecia universalis describes all body hair loss (FIGURE 2 – 24). Loss of scalp hair may occur in virilizing female, hypothyroidism and severe iron deficiency anaemia. Thin rough hairs may occur in uremia.



A B C D

FIGURE 2 – 24. Patients with alopecia areata (A) and with alopecia universalis (B, C, and D). There is loss of eye lashes and eye brows (D).

THE FACE

A specific diagnosis can sometimes be made by inspecting the face, its appearance giving a clue to the likely diagnosis. Some facial characteristics are so typical of certain diseases that they immediately suggest the diagnosis and are called THE DIAGNOSTIC FACIES. These (FIGURES 2 – 25 and 2 – 26) are:

1	Hyperthyroidism	Patient has excessive emotional movement with eye lid retraction and exophthalmos or ophthalmoplegia, most commonly affects female
2	Hypothyroidism	Patient has no emotions with pallor and puffiness of face, most commonly affects female
3	Acromegaly	Patient has broad face with prominence of the supraorbital ridges, lower jaw and cheeks
4	Cushing's syndrome	Patient has moon face, acne, and hirsutism with thinning of skin
5	Down's syndrome	Patient has flat face with slanting eyes, epicanthic folds, snub nose, protruding tongue and unusual ears as well as round head
6	Addison's disease	Marked pigmentation found on the face and on the exposed areas of the skin e.g. fingers, palmar creases, elbows, and scars
7	Myotonia dystrophica	Frontal baldness (the patient may be wearing a wig), the expressionless triangular facies (monk-like appearance), atrophy of the temporalis muscle and partial ptosis.
8	Neurofibromatosis	Patient has multiple skin-colored and pinkish papules and nodules on face
9	Sturge-Weber syndrome	It is characterized by an angiomatous malformation on the face (portwine stain), epilepsy and mental deficiency
10	Nephrotic syndrome	Puffy pale face with periorbital bagginess
11	Superior vena cava obstruction	The patient has a non pulsatile distension of neck veins, edema and cyanosis of the head, neck, hands and arms
12	Myasthenia gravis	Patient has dropping of the upper eyelids and chin that gives rise to a half closed eye and opened mouth with sneering smile as well as weakness of the muscles of the face with loss of facial expression.
13	Anorexia nervosa	Facial appearance suggests undernutrition but patient insists that they are eating well even many believe that she is overweight
14	Parkinson's disease	Mask face with evidence of poor muscular mobility

15 Pickwickian syndrome	Patient is characterized by a short neck and facial plethora due to morbid obesity
16 Bell's palsy	Paralysis of the upper and lower parts of the face with loss of the forehead wrinkles and the nasolabial fold on the affected side. The corner of the mouth appears to droop.
17 Hippocratic	This is seen in advanced peritonitis. The eyes are sunken, temples collapsed, nose is pinched with crusts on the lips and the forehead is clammy
18 Marfanoid	In Marfan's syndrome, the limbs are long in relation to the length of the trunk, and the arm-span exceeds height. Additional features include long slender fingers (arachnodactyly), narrow feet, a high-arched palate, upward dislocation of the lenses of the eyes, and cardiovascular abnormalities such as mitral valve prolapse, and dilatation of the aortic root with aortic regurgitation.
19 Turner's syndrome	Typical features include short stature, webbing of the neck, small chin, low-set ears, low hairline, short fourth finger, increased carrying angle at the elbows and widely spaced nipples ('shield-like chest').
20 Uremic	Earthy pigmented face with lemon-yellow coloration of the skin (uremic complexion), and bruising and excoriation secondary to pruritus. These patients are often anemic and have a urine-like smell on the breath (uremic fetor). Nail changes include half and half nail, leukonychia, and Beau's lines. There may be a surgically created arteriovenous (AV) fistula at the wrist or elbow to allow vascular access for hemodialysis.
21 Virile facies	Virilization refers to a condition in which androgen levels are sufficiently high to cause hirsutism, deepening of the voice, breast atrophy, increased muscle bulk, clitoromegaly, and increased libido.

FIGURE 2 – 25. Characteristic diagnostic facies including hyperthyroidism (A), hypothyroidism (B), acromegaly (C), Cushing syndrome (D), Down syndrome (E), Addison's disease (F), myotonia dystrophica (G), and neurofibromatosis (H).

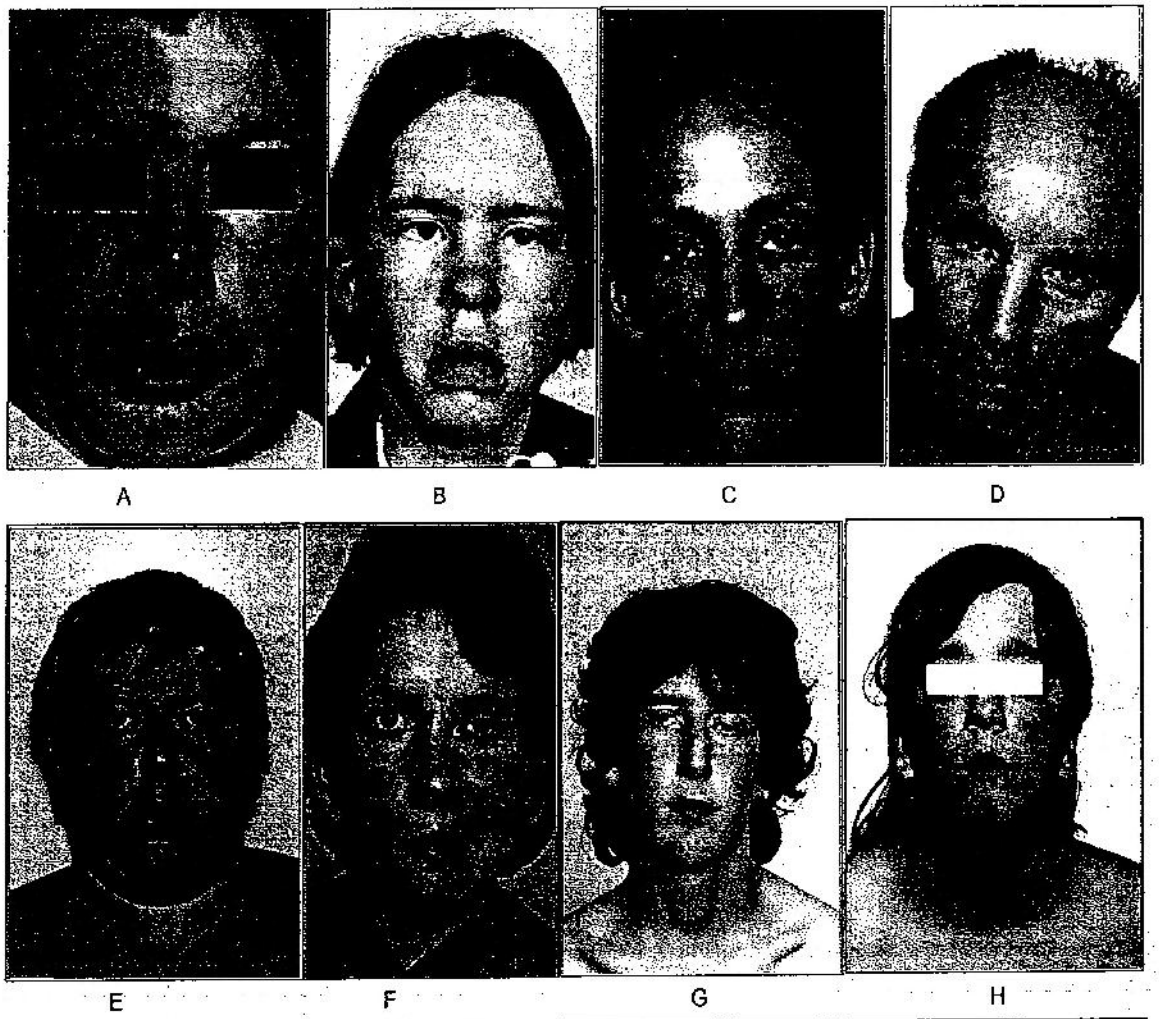


FIGURE 2 – 26. Characteristic diagnostic facies including Sturge-Weber syndrome (A), Myasthenia gravis (B), Anorexia nervosa (C), Parkinson's disease (D), Pickwickian syndrome (E), Bell's palsy (F), Marfan's syndrome (G), and Turner's syndrome (H).

Apart from the characteristic diagnostic facies, there are several other important abnormalities that must be looked for in the face. Frontal bossing may occur in Paget's disease (FIGURE 2 – 27), acromegaly, rickets, thalassemia, sickle cell anemia, and congenital hydrocephalous. Supraorbital ridges are enlarged in acromegaly. In temporal arteritis, temporal artery is palpable and visible (FIGURE 2 – 27). Congested face occurs in alcoholics and in case of polycythemia. Red raised eruptions on the bridges of the nose extending to the cheek commonly in female due to systemic lupus erythematosus (SLE). This is called batwing or butterfly rash (FIGURE 2 – 27). Malar rash may also occur in dermatomyositis, erysipelas, or rosacea. Xanthelasma is a deposition of lipid in the eye lids (commonly upper) and occurs either idiopathically or due to hyperlipidemia (FIGURE 2 – 27). Pale cheeks may be racial normal finding or may occur in anemia or hypopituitarism. Pale and puffy cheeks occur in nephrotic syndrome. Malar flush which is a bright circumscribed flush occurs in mitral stenosis, carcinoid syndrome, pheochromocytoma, and in patients treated with niacin or steroid. Bluish tint of the cheeks may suggest congestive heart failure. Causes of enlarged parotid glands are shown in BOX 2 – 5.

BOX 2 – 5. Causes of parotid gland swelling

Infection	Tumours	Other
Mumps Bacterial (post-operative)	Benign: pleomorphic adenoma (95% of cases) Malignant: carcinoma	Calculi Sjögren's syndrome

Tender bilateral or unilateral parotid swelling occurs in Mumps (FIGURE 2 – 27); unilateral swelling and reddening of the skin may suggest acute parotitis; persistent enlargement accompanied by dry tearless eyes in Sjögren's syndrome (FIGURE 2 – 23); unilateral irregular painless swelling may suggests mixed parotid tumor (FIGURE 2 – 27); bilateral enlargement with congested face occurs in alcoholic person.

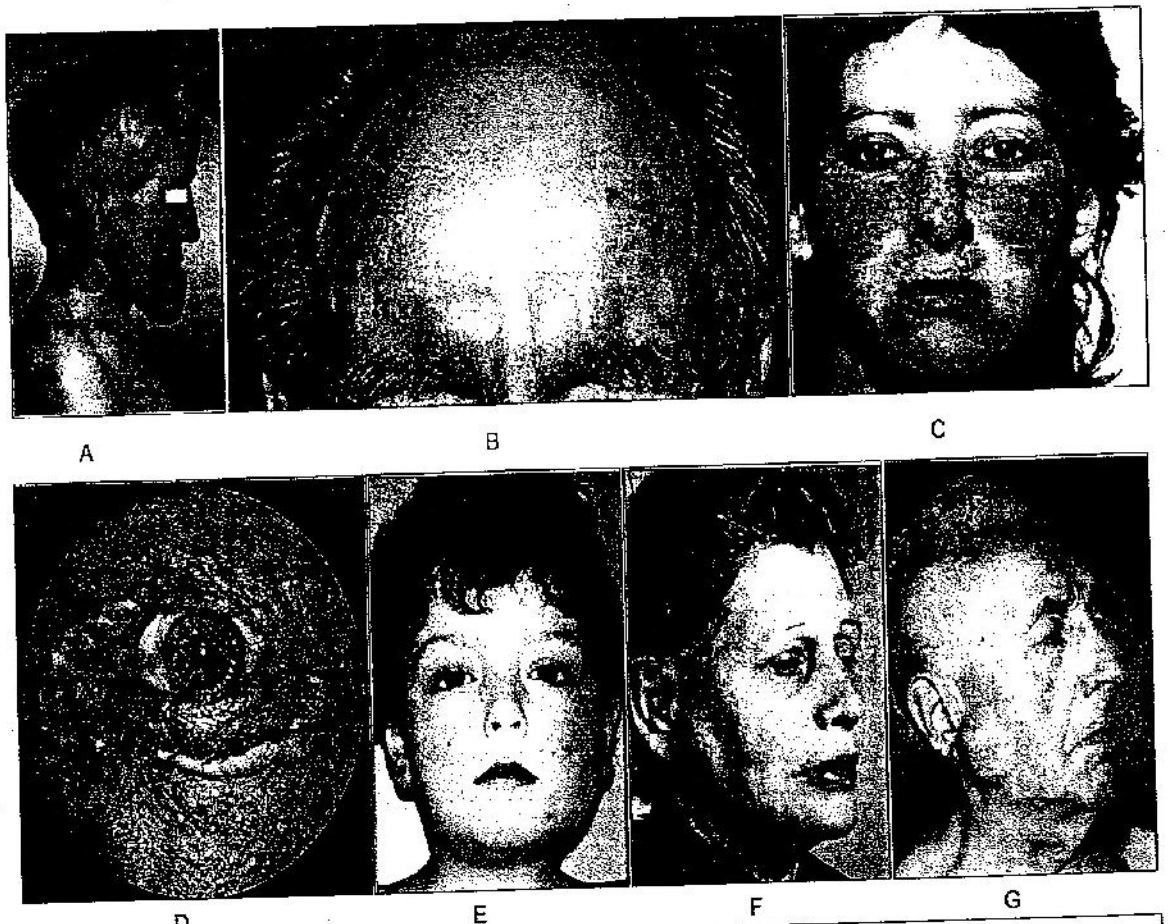


FIGURE 2 – 27. Various facial abnormalities including frontal bossing in Paget's disease (A), visible temporal artery in temporal arteritis (B), systemic lupus erythematosus rash (C), Xanthelasma (D), parotid enlargement in Mumps (E), Sjögren's syndrome (F) with parotid gland enlargement, and mixed parotid tumor (G).

THE EYES

The initial inspection should focus on each part of the external eye namely: the orbit, the eyebrows, the eyelashes, the eyelids; the two canthi where the lower and upper eyelids meet; the conjunctiva; the cornea; the iris; and the lens (FIGURE 2 – 28). ANEMIA (FIGURE 2 – 29) is simply defined as a reduction in the number of circulating erythrocytes and is recognized in the laboratory when a patient's hemoglobin level or hematocrit is reduced below the normal range. Anemia is manifested clinically as pallor. The pallor of anaemia is best seen in the mucous membranes of the conjunctivae, lips and tongue, in the nail beds, and in the palmar creases. JAUNDICE (FIGURE 2 – 29) is a yellowish discoloration of the skin, the mucous membrane and sclerae (which have a particular affinity for bilirubin due to their high elastin contents) caused by increased serum bilirubin concentration that are deposited in the tissue. The candidate should examine the eyes simultaneously for anemia and jaundice using the following steps (FIGURE 2 – 30):

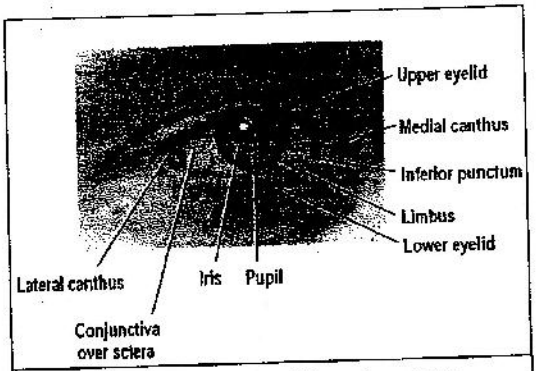


FIGURE 2 – 28. Anatomy of the external eye.

1. STEP I Ask the patient to turn his/her head into natural light (in ward, the day light is obtained through windows) because jaundice that is obvious in daylight may be undetected in artificial light.
2. STEP II Raise the upper eye lid using the thumb of your left hand to inspect upper sclera meanwhile the patient is looking to your right hand moving downwards.
3. STEP III Ask the patient to look upward while you draw the lower eye lid downward to inspect blood vessels of conjunctiva.

FIGURE 2 – 29. Yellowish discoloration of the sclera (A) and pale conjunctive (B).

Another useful site to look for pallor and jaundice is the sublingual mucosa after asking the patient to open his/her mouth and raise tongue. The palmar creases turned pale (in comparison with your palm) when the hemoglobin is less than 7 g/dL and discolored yellow in jaundice. In long standing jaundice, a distinct greenish colour develops in sclerae and skin owing to the presence of biliverdin. Scratch marks may be prominent in obstructive jaundice due to pruritus. Normal serum bilirubin level is 1 mg/dL. Jaundice becomes clinically evident when serum bilirubin exceeds 3 mg/dL. The differential diagnosis for yellowing of the skin is limited. In addition to jaundice, it includes carotenoderma, the use of the drug quinacrine, and excessive exposure to phenols. Carotenoderma (hypercarotenemia) is the yellow color imparted to the skin by the presence of carotene; it occurs in healthy individuals who ingest excessive amounts of vegetables and fruits that contain carotene, such as carrots, leafy vegetables, squash, peaches, and oranges. Unlike jaundice, where the yellow coloration of the skin is uniformly distributed over the body, in carotenoderma the pigment is concentrated on the palms, soles, forehead, and nasolabial folds. Carotenoderma can be distinguished from jaundice by the sparing of the sclerae. Quinacrine causes a yellow discoloration of the skin in 4-37% of patients treated with it. Unlike carotene, quinacrine can cause discoloration of the sclerae.

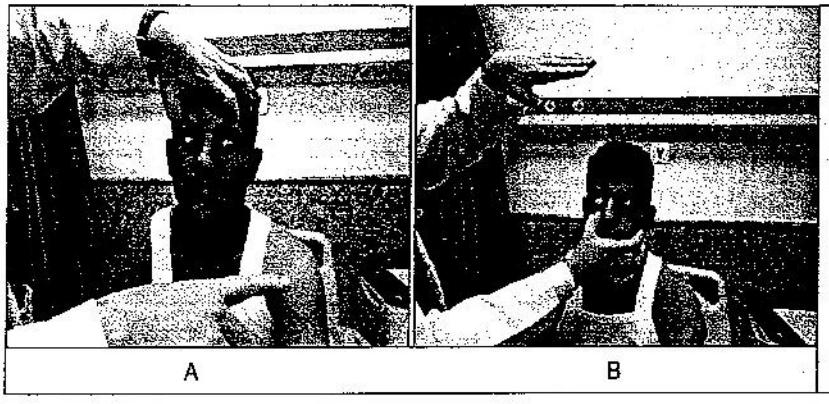


FIGURE 2 – 30. The technique for examining the sclera looking for jaundice (A) and for examining the conjunctiva looking for pallor (B).

When bilirubin level is less than 1 mg/dl the person had no jaundice; bilirubin level 1 – 3 mg/dl indicates biochemical jaundice while bilirubin level more than 3 mg/dl indicates clinical jaundice.
Do not confuse the diffuse yellow sclerae of jaundice with the small yellowish fat pads (pinguecula) sometimes seen at the periphery of the sclerae. Other than anemia and jaundice the candidate should check the eyes for other clinical signs (FIGURE 2 – 31). Bluish discoloration of the sclera occurs in iron deficiency anaemia, rheumatoid arthritis, hyperthyroidism, Ehlers-Danlos syndrome, and osteogenesis imperfecta. Subconjunctival hemorrhages are the result of the rupture of small vessels from increased intravascular pressure during severe coughing and in patients with hypertension. Cataracts of the lens develop in people over the age of 60 years and called senile cataract but the process may appear under the age of 50 years because of systemic disorders. Kayser-Fleischer ring describes a yellowish to brown thin band of copper deposit at the upper and lower limbus. It is associated with Wilson's disease. Corneal arcus (Arcus lipidus) which is a white ring near the outer margin of the cornea; in the elderly it is called arcus senilis which may be of no clinical significance but in young it is called arcus juvenilis and is indicative of hypercholesterolemia. Raccoon eyes describe bluish discoloration of the periorbital area from the extravasated blood after a fracture of the anterior cranial fossa. Look also for eye signs of the thyrotoxicosis; lid retraction and exophthalmos. Ptosis (dropping of the upper eye lid) and proptosis (bulging of the eye ball) should also be looked for. Lack of the outer third of eye brows may occur in leprosy, scleroderma, and hypothyroidism. Pupillary dilation, mydriasis, is seen in oculomotor nerve palsy, Holmes Adie syndrome, midbrain lesion, pheochromocytoma, or with use of sympathomimetics or anticholinergics. Pupillary constriction, miosis, is seen in Horner's syndrome, Argyll Robertson syndrome, pontine lesion, or poisoning with opiate and organophosphorus compounds.

FIGURE 2 – 31. Some clinical signs in eye disorders including bluish discoloration of the sclera (A), Kayser Fleischer ring (B), partial ptosis of the left eye (C), cataract of the lens (D), proptosis of the eyes (E), raccoon eye (F), complete ptosis of the right eye (G), exophthalmos of the eye (H), miosis of the left eye (I), corneal arcus (J), and subconjunctival hemorrhage (K).

Kayser-Fleischer ring first appears as a superior crescent, then develops inferiorly and ultimately becomes circumferential. Kayser-Fleischer rings are present in more than 99% of patients with neurologic/psychiatric forms of Wilson disease and in only 30–50% of Wilson patients with hepatic or presymptomatic state.

WHAT ARE THE EYE SIGNS IN PATIENT WITH WILSON DISEASE?
Bilirubin is deposited in the sclerae producing jaundice.
Copper deposition produces Kayser-Fleischer ring
Copper can also accumulate in the lens and produce "sunflower" cataracts.

THE NOSE

Anatomical parts of the nose are shown in FIGURE 2 – 32. Nasal abnormalities (FIGURE 2 – 33) are commonly seen during clinical practice. Beaking of the nose is seen in systemic sclerosis; saddle-nose is a depression of the nasal bridge due to nasal septum destruction and occur in relapsing polychondritis, Wagener's granulomatosis, leprosy, and congenital syphilis; large nose may occur in acromegaly; rhinophyma is a red enlarged and bulbous nose due to rosacea; nasal polyps can be seen on the nasal septum and are associated with asthma; nasal discharge can be seen in upper respiratory tract infection.

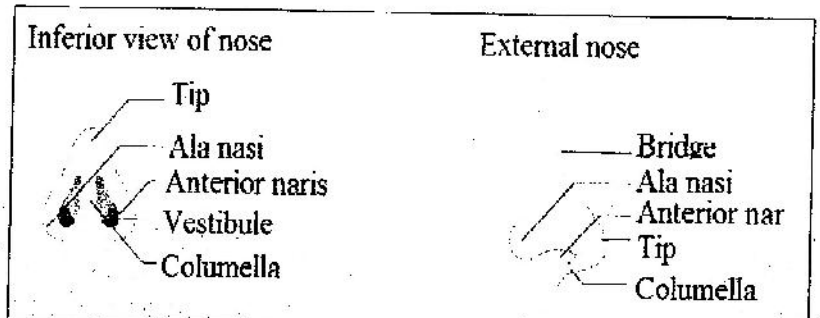


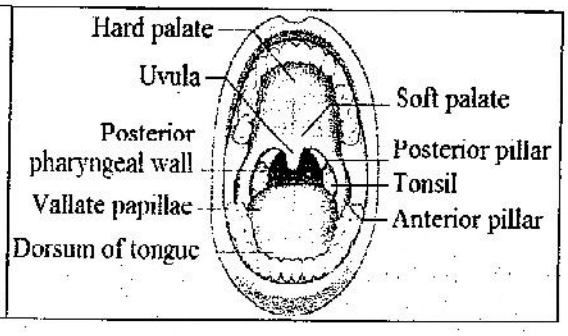
FIGURE 2 – 32. Anatomy of the nose.

FIGURE 2 – 33. Some clinical nasal signs including bluish beaked nose (A), saddle nose (B), large nose (C), and rhinophyma (D).

THE MOUTH

Mouth examination should include valuable approach to the anatomical parts provided in FIGURE 2 – 34 and also for breath smell.
The clinical signs (FIGURE 2 – 35) can be elicited when the candidate conduct the examination in the following steps:

FIGURE 2 – 34. Anatomical parts of the mouth.



1 STEP I Inspect the mouth in the resting position

The mouth is often kept open in Down syndrome with the tongue partially protruding through it; asymmetry of the face and the angles of the mouth may suggest facial nerve palsy; circumoral wrinkling (radial furrowing or vertical lines) occurs in systemic sclerosis. An acute inflammation of the lips (cheilitis or stomatitis) with painful fissuring and scaling at the angles of the mouth (angular cheilitis or angular stomatitis) is caused by dribbling from ill-fitting dentures or deficiency states of iron or vitamin B_{12} and may not be obvious unless specially looked for. In angioneurotic oedema, the subcutaneous tissue rather than the dermis is swollen, usually affecting the lips, mouth, eyes and genitalia. Blue colored lips indicate cyanosis. More than 95% of patients with Peutz-Jeghers syndrome have a characteristic pattern of brown black melanin spots on the lips, buccal mucosa, and skin.

2 STEP II Ask the patient to open his/her mouth

Microstomia (reduced opening of the mouth) may suggest systemic sclerosis.

3 STEP III Check the outer and the inner surfaces of both upper and lower lips

Oral ulcer is located in any part of the mouth and may be idiopathic or due to (BOX 2 – 6) other disorders.

4 STEP IV Check the outer and lateral surfaces of the tongue and ask the patient to put out and then to raise his/her tongue to see the sublingual structures

Fasciculation (irregular ripples or twitching) of the tongue occurs in lower motor neurone disorders. Check then the tongue for tremor that may occur in Parkinsonism, thyrotoxicosis and in anxiety. Fasciculation is checked whilst the tongue is inside the mouth while tremor is evident when the patient put his/her tongue out of the mouth. Enlargement of the tongue (macroglossia) may occur in acromegaly, amyloidosis or tumour infiltration. Bluish discoloration is a good clue for central cyanosis. Dry brown tongue may suggest uremia or acute intestinal obstruction. White patches that may be scraped off the tongue are due to candida (oral thrush) and common predisposing factors include inhaled steroids or immune deficiency e.g. chemotherapy, or diabetes mellitus. Glossitis is a smooth reddened tongue due to atrophy of the papillae (depapillated tongue). It is common in alcoholics, in nutritional deficiencies of iron, folate and vitamin B12. Glossitis may cause a burning sensation over the tongue but usually a painful tongue is a symptom of anxiety or depression. Hairy leukoplakia is a white plaque occurring frequently on the lateral surfaces of the tongue bilaterally in immunosuppressed persons. The sublingual area is good site suggesting anemia, jaundice, or cyanosis.

5 STEP V Check the remaining structures of the oral cavity including gums, teeth, tonsils, and internal mucosa

The inside of the mouth and the tongue should be inspected for telangiectasia (Osler-Weber-Rendu syndrome) and pigmentation (Addison's disease). Gum hyperplasia occurs in pregnancy, acute myeloid leukemia (M4), and with drugs e.g. calcium channel blockers, phenytoin, and ciclosporine. Dental caries may be valuable in infective endocarditis. High arched palate suggests Marfan's syndrome.

6 STEP Check the smell of breath

Breath smell gives certain suggestion when notified. Halitosis (bad odor) occurs in pyorrhea, pyloric obstruction, gastrocolic fistula, or suppurative lung disease e.g. lung abscess and bronchiectasis; alcoholic smell is present in alcoholics; acetone breath smell may be noticed in diabetic ketoacidosis; ammoniacal (fishy) smell is found in uremia (uremic fetor); a sweet musty smell is present in liver failure (hepatic fetor); smell of certain drugs e.g. paraldehyde may also be noted and of course the stale cigarette smell of the patient who smokes.

BOX 2 – 6. Causes of oral ulceration

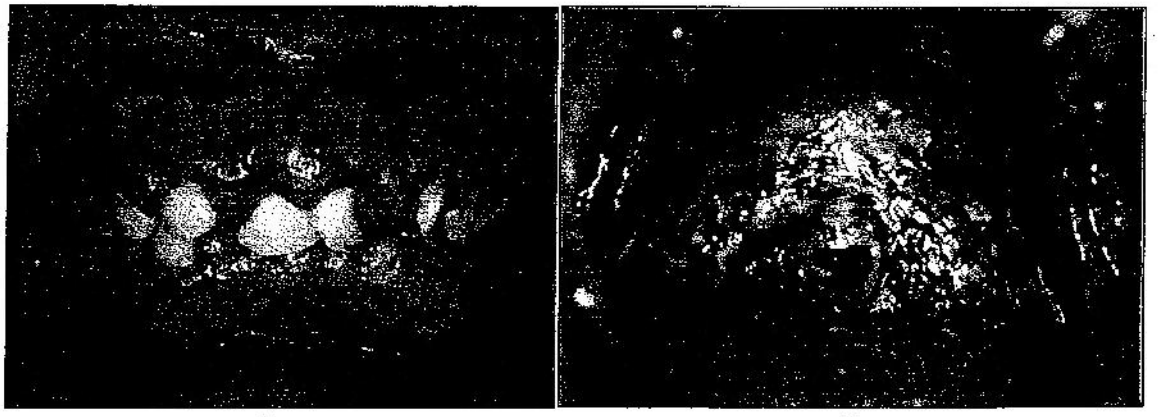
Aphthous		Drugs	
Idiopathic	Premenstrual	Cytotoxic drugs	Drug hyper-sensitivity
Infection		Dermatological diseases	
Viral e.g. herpes simplex	Bacteria e.g. syphilis Fungus e.g. Candida	Lichen planus Pemphigus	Pemphigoid
GIT diseases		Systemic diseases	
Crohn's disease	Coeliac disease	Behçet's syndrome	SLE
Neoplasia		Vasculitis	
Kaposi's sarcoma	Carcinoma Leukemia	Wegener's granulomatosis	

CLINICAL CLUE

The clinical findings in the face of a patient with systemic sclerosis may include any one of taut skin, radial furrowing, microstomia, multiple telangiectasia, beaked nose, pallor (due to gastrointestinal blood loss), cyanosis (due to fibrosing alveolitis or pulmonary hypertension), and jaundice (due to hemolysis from blood transfusion or liver secondaries from esophageal or bowel adenocarcinoma)

A B C

D E F



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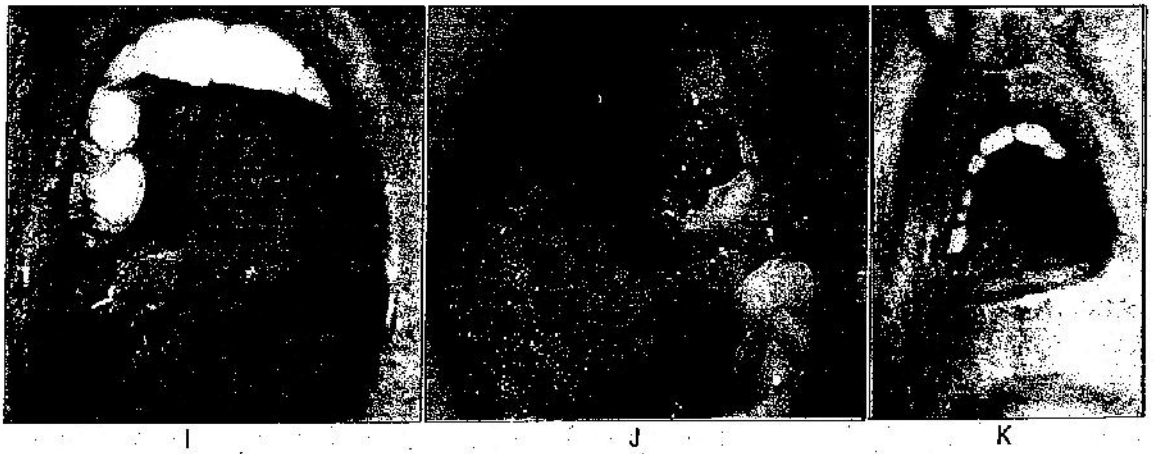

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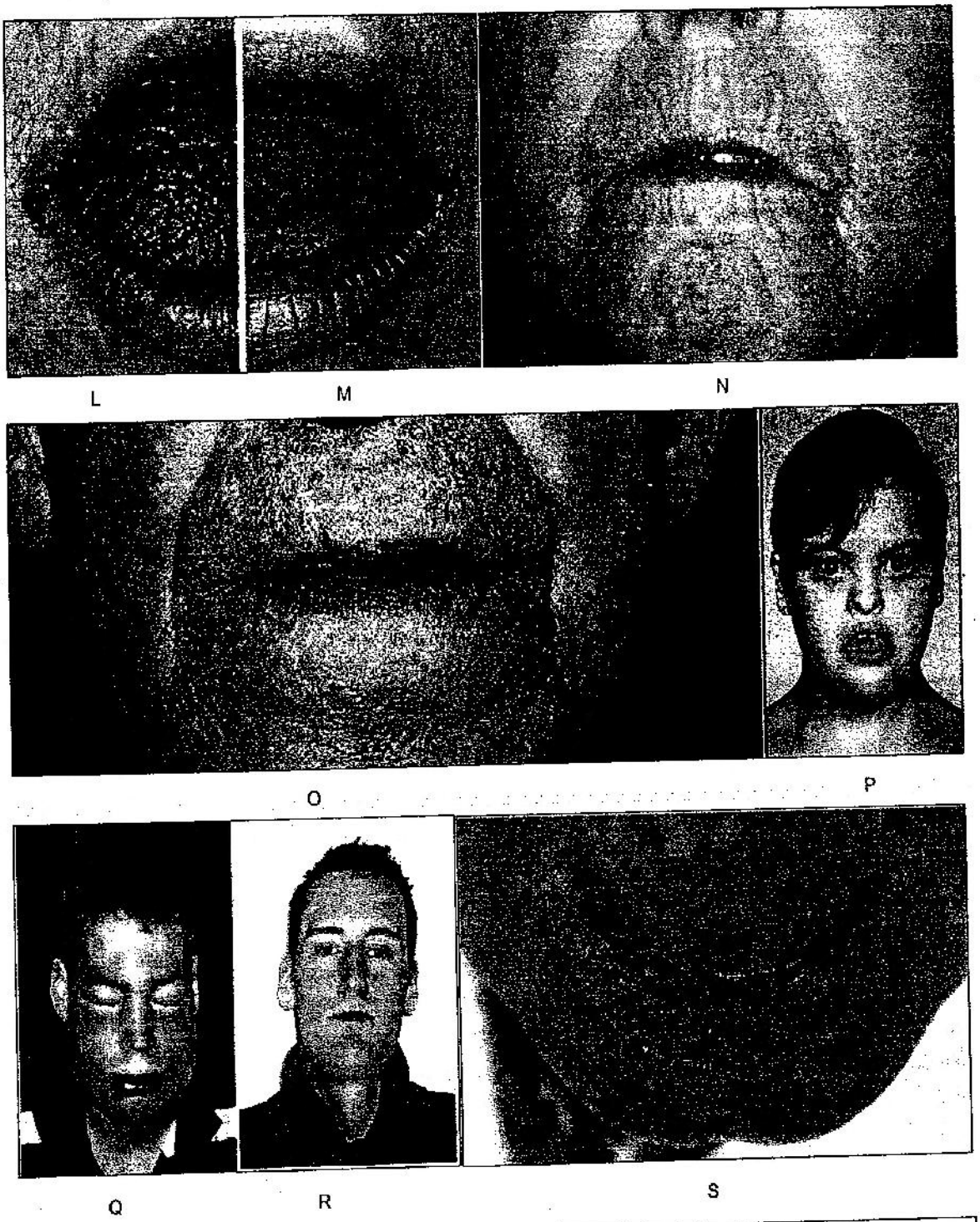


FIGURE 2 – 35. Some clinical oral conditions including microstomia with multiple tongue telangiectasia (A), right facial nerve palsy with deviation of the mouth to the left side (B), large tongue due to acromegaly (C), multiple shallow oral ulceration (D), solitary labial ulcer (E), hairy leukoplakia (F), gingival hyperplasia (G), oral thrush due to candidiasis (H), pigmented spots due to Addison disease (I), dental caries (J), high-arched palate due to Marfan's syndrome (K), angular stomatitis with depapillated glossitis before treatment (L), and after treatment (M), radial furrowing of scleroderma (N), cyanosis of the lips (O), opened mouth and partially protruded tongue in patient with Down syndrome (P), angioneurotic edema before treatment (Q), and after treatment (R), and brown black melanin spots in patient with Peutz-Jeghers syndrome (S).

Cyanosis refers to a bluish discoloration of the skin and mucous membrane resulting from an increased quantity of reduced hemoglobin or of hemoglobin derivatives in the small blood vessels of those areas. In general, cyanosis becomes apparent when the concentration of reduced hemoglobin in capillary blood exceeds 5 g/dl. Cyanosis may be subdivided into central and peripheral types (BOX 2 – 7)

BOX 2 – 7. Types and causes of cyanosis

Central cyanosis	Peripheral cyanosis
Found in the mucous membrane and skin	Found In the skin only
Hot extremities	Cold extremities
Clubbing of finger may be present	No clubbing of finger
This type of cyanosis results from 1. Cyanotic congenital heart disease 2. Respiratory failure 3. Heart failure 4. Abnormal hemoglobin derivatives e.g. methemoglobin and sulfhemoglobin	This type of cyanosis results from 1. Exposure to cold 2. Heart failure 3. Arterial obstruction 4. Venous obstruction

A type of central cyanosis is called differential cyanosis. In patent ductus arteriosus complicated by Eisenmenger syndrome, the severe pulmonary vascular disease results in reversal of the blood flow through the ductus; unoxygenated blood is shunted to the descending aorta; and the toes, but not the fingers, become cyanotic and clubbed, a finding termed differential cyanosis and differential clubbing.

A type of peripheral cyanosis is called acrocyanosis. In this condition, there is arterial vasoconstriction and secondary dilation of the capillaries and venules with resulting persistent cyanosis of the hands and, less frequently, the feet. Cyanosis may be intensified by exposure to a cold environment. Acrocyanosis may be categorized as primary or secondary to an underlying condition. In primary acrocyanosis, women are affected much more frequently than men, and the age of onset is usually <30 years. Generally, patients are asymptomatic but seek medical attention because of the discoloration. Pain, ulcers, and gangrene do not occur. Examination reveals normal pulses, peripheral cyanosis, and moist palms. The disorder can be distinguished from Raynaud's phenomenon because it is persistent and not episodic, the discoloration extends proximally from the digits, and blanching does not occur. Ischemia secondary to arterial occlusive disease can usually be excluded by the presence of normal pulses. Central cyanosis and decreased arterial oxygen saturation are not present. Secondary acrocyanosis may result from connective tissue diseases, atheroembolism, antiphospholipid antibodies, cold agglutinins, or cryoglobulins.

Examination for cyanosis should follow the following steps:

1 STEP I Inspect the lips, tongue and sublingual mucosa; cyanosis in these areas is usually central. If spared with only involvement of extremities peripheral cyanosis is likely the cause.

2 STEP II Touch both hands of the patients; warm hands (with or without digital clubbing) indicate central cyanosis while cold hand without clubbing indicates peripheral cyanosis. Cyanosis of the lips, tongue, and extremities with cold hands indicates central and peripheral cyanosis.

3 STEP III Massage or gentle warming of a cyanotic extremity will increase peripheral blood flow and abolish peripheral, but not central cyanosis.

IMPORTANT NOTE

Clinical differentiation between central and peripheral cyanosis may not always be simple, and in conditions such as cardiogenic shock with pulmonary edema there may be a mixture of both types. Thus one can comment on cyanosis as either central or peripheral or peripheral and central.

HAD YOU SEEN A CLINICAL CONDITION WITH?

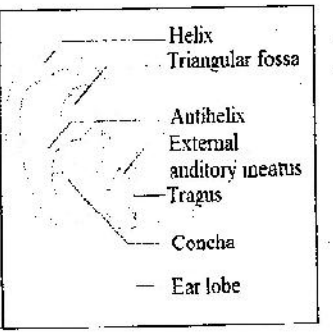
1. Pallor, jaundice, and cyanosis.
2. Yellowish discoloration involving single eye.
3. Single sclera colored black and the other colored yellow.

1. Patient with G6PD who had taken dapson. He may develop anemia and jaundice due to hemolysis and may develop central cyanosis due to methemoglobinemia induced by the drug.
2. This implies local absorption of eye hematoma.
3. This implies melanoma (black) with metastasis to the liver.

THE EXTERNAL EAR

Anatomy of the external ear is shown in FIGURE 2 - 36. Abnormal clinical findings (FIGURE 2 – 37) may include congenital malformation or absence of all or part of the ear is rare but clinically significant because of its association with deafness and facial asymmetry; otitis externa is the most frequent disorder involving the external auditory meatus and often extends to the pinna; gouty tophi may also be noticed in the ear; a bluish discoloration may suggest cyanosis. Various skin tumours such as squamous cell carcinoma sometimes arise on the pinna.

FIGURE 2–36. Anatomy of the external ear.



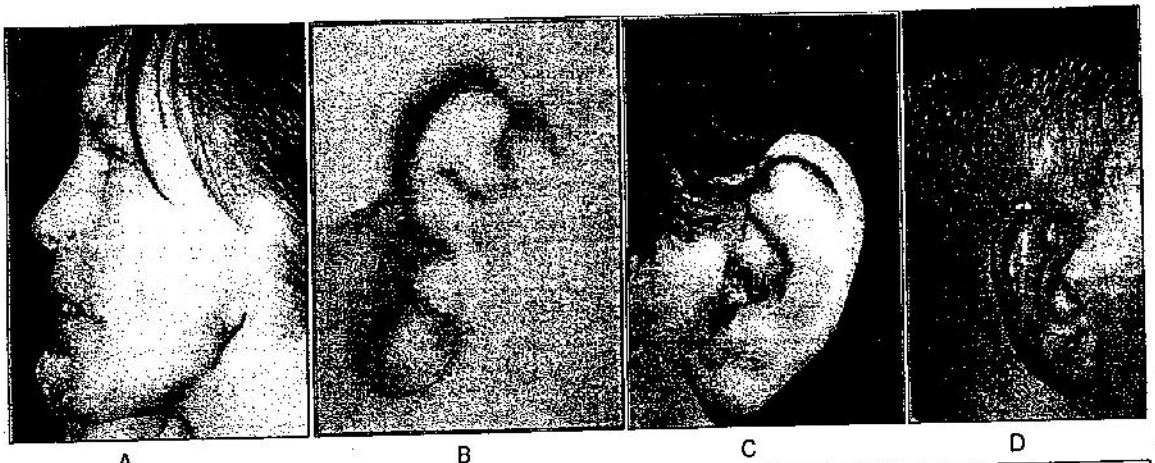


FIGURE 2 – 37. Some clinical conditions affecting the external ear include congenital absence of the external ear (A), congenital abnormality in the form of cauliflower external ear (B), otitis externa with visible discharge (C), and gouty tophus involving the external ear.

THE NECK

For descriptive purposes, each side of the neck is divided into two triangles (FIGURE 2 – 38) by the sternomastoid (sternocleidomastoid) muscle. The anterior triangle is bounded above by the mandible, laterally by the sternomastoid, and medially by the midline of the neck. The posterior triangle extends from the sternomastoid to the trapezius and is bounded below by the clavicle. A portion of the omohyoid muscle crosses the lower portion of the posterior triangle and can be mistaken by the uninitiated for a lymph node or mass. Deep to the sternomastoids run the great vessels of the neck: the carotid artery and internal jugular vein (FIGURE 2 – 38). The external jugular vein passes diagonally over the surface of the sternomastoid.

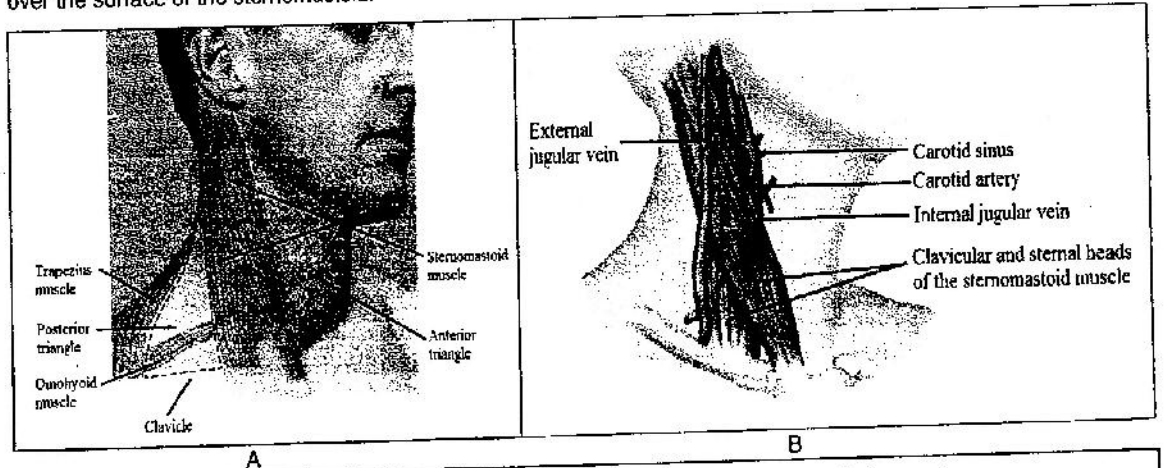


FIGURE 2 – 38. Triangles of the neck (A) and the anatomical relationship of the cervical vessels.

Full assessment of the neck should include the following steps of examination:

1. STEP I Head and neck lymph node examination.
2. STEP II Examination of the thyroid gland. During general examination the thyroid gland is examined only by inspection for any swelling and palpation in the resting state. If you find an abnormality involving the thyroid gland you should take permission from the examiner to wholly examine the thyroid glands.
3. STEP III Examination of the position of the trachea
4. STEP IV Examination of the blood vessels
5. STEP V Other findings e.g. masses, long neck or short neck

HEAD AND NECK LYMPH NODES EXAMINATION

The lymph node groups of the head and neck are shown in FIGURE 2 – 39. The candidate must decide whether the lymphadenopathy is a normal or abnormal finding. Soft, flat, submandibular nodes (< 1 cm) are often palpable in healthy children and young adults, and healthy adults may have palpable inguinal nodes of up to 2 cm, which are considered normal.

Generalized lymphadenopathy has been defined as the involvement of three or more noncontiguous lymph node areas.

Lymph nodes are considered abnormal and lymph node biopsy is indicated if the age of the patient > 40 years, supraclavicular location, node size >2.25 cm^2, hard texture, and lack of pain or tenderness.

When supraclavicular lymph nodes are enlarged by metastasis, they are called (Virchow's node) and its presence is called (Troisier's sign).

The technique of lymph nodes examination is performed according to the following steps (FIGURE 2 – 40):

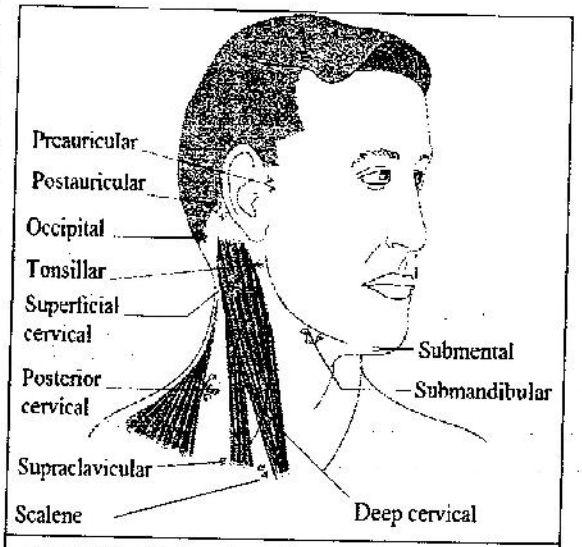


FIGURE 2 – 39. Location of head and neck lymph nodes.

1. STEP I Examine the cervical nodes with the patient sitting.
2. STEP II From behind, examine the submental, submandibular, preauricular, tonsillar, superficial cervical and deep cervical nodes in the anterior triangle of the neck. Palpate the lymph nodes using the fingers of each hand and compare with the nodes on the contralateral side. For the cervical lymph nodes, palpate each side in turn and ask the patient to tilt his head to the same side of examination.
3. STEP III Palpate for the scalene nodes by placing your index finger between the sternocleidomastoid muscle and clavicle. Ask the patient to tilt his head to the same side of examination
4. STEP VI With the patient's shoulders slightly shrugged, feel in the supraclavicular fossa and at the base of the sternomastoid muscle for the supraclavicular nodes.
5. STEP V From the front of the patient, palpate the posterior triangles, up the back of the neck for the posterior cervical, postauricular and occipital nodes.

WHEN YOU FIND A LYMPH NODE THE PRESENTATION SHOULD INCLUDE (MNEMONIC: SPACES)

Size	Nodes < 1.0 cm^2 in area (1.0 cm x 1.0 cm or less) are almost always secondary to benign, nonspecific reactive causes. A lymph node size of 2 cm^2 is abnormal until proven otherwise.
Position (the site)	Important diseases such as both acute and chronic infections and metastatic carcinoma will cause localized lymphadenopathy depending on the site of primary pathology.
Attachments	Nodes that are fixed to surrounding tissue are highly suspicious of malignancy.
Consistency	Malignant lymph nodes feel unusually firm or hard and irregular. Enlarged nodes secondary to infection may feel soft.
Extrafeatures (delimitation, number, and tenderness)	Delimit the lymph nodes as discrete or matted together. Nodes involved by lymphoma tend to be discrete while matted glands may occur in tuberculous lymphadenopathy. Define the number of the lymph nodes. Tenderness is found when the capsule is stretched during rapid enlargement, usually secondary to an inflammatory process. Some malignant diseases such as acute leukemia may produce rapid enlargement and pain in the nodes
Shape	Determine the node shape e.g. ovoid, round, or spherical

FIGURE 2 – 40. The clinical examination of the anterior cervical lymph nodes (A), scalene lymph nodes (B), and the supraclavicular (C) lymph nodes. Examination of the posterior cervical, postauricular, and occipital lymph nodes is conducted from the front (D).

Causes of lymphadenopathy are shown in BOX 2 – 8

BOX 2 – 8. Causes of lymphadenopathy

Infectious diseases	Other disorders
• Viral e.g. EBV, CMV, HIV • Bacterial e.g. streptococci, staphylococci, brucellosis, tuberculosis, syphilis • Parasitic e.g. toxoplasmosis, leishmaniasis • Fungal e.g. histoplasmosis, coccidioidomycosis	Sarcoidosis Familial mediterranean fever
Immunologic diseases	**Malignant diseases**
• Rheumatoid arthritis • Systemic lupus erythematosus • Sjögren's syndrome • Mixed connective tissue disease	• Hematological e.g. lymphomas, lymphocytic leukemia, amyloidosis • Metastatic from numerous primary sites
Endocrine diseases	**Drug hypersensitivity**
Hyperthyroidism	Phenytoin, hydralazine, allopurinol, gold, carbamazepine

EXAMPLES

There is enlarged tonsillar lymph node that is tender, spherical in shape, about two by two cm^2, rubbery in consistency, and not fixed to the skin

What is the diagnosis?
Non- specific Inflammatory lymphadenopathy

Enlarged posterior cervical lymph nodes that are three in number, ovoid in shape, largest one is two by two cm^2, matted together and non-tender

What is the diagnosis?
Tuberculous lymphadenopathy

Enlarged five superficial lymph nodes, about three by three cm², discrete, symmetric, non tender, mobile and rubbery firm in consistency

What is the diagnosis?
The diagnosis is lymphoma

There is enlarged left supraclavicular lymph node, two by two cm², non-tender, smooth surface, hard in consistency, and non-movable because of fixation to surrounding tissues

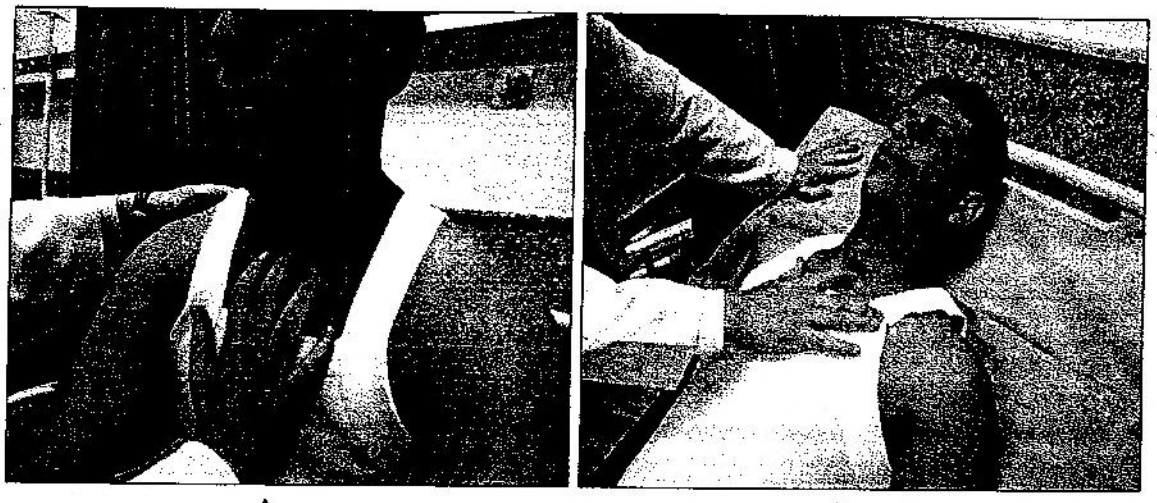
What is the diagnosis?
The diagnosis is secondary metastasis from primary tumor

CLINICAL CLUE

Lymph-node tuberculosis presents as painless swelling of the lymph nodes, most commonly at posterior cervical and supraclavicular sites, a condition historically referred to as scrofula.

EXAMINATION OF THE POSITION OF THE TRACHEA

It is usually conducted by gently introducing the tip of the index finger into the suprasternal notch exactly in the midline and press gently against the trachea (FIGURE 2 – 41). The middle finger may be used to localize the trachea with additional placement of the right ring finger on the attachment site of the sternomastoid muscle with the clavicle and index finger on the other side. If the trachea is deviated, the finger will slip partially or completely past it on the side opposite to the displacement.



A B

FIGURE 2 – 41. Examination of the position of the trachea

CLINICAL CLUE

Examination of the thyroid gland and other central and peripheral masses is dealt with in other chapter. Such an examination is requested from fourth medical student and elder. It should not be part of clinical training for third medical students.

EXAMINATION OF THE BLOOD VESSELS

CAROTID ARTERY

The patient's right carotid artery is examined by gentle compression against the transverse processes of the cervical vertebrae with the candidate's left hand (FIGURE 2 – 42). Try not to compare right with left carotid artery at the same time as this may induce syncope. Carotid pulse is used to describe the volume and character of blood flow.

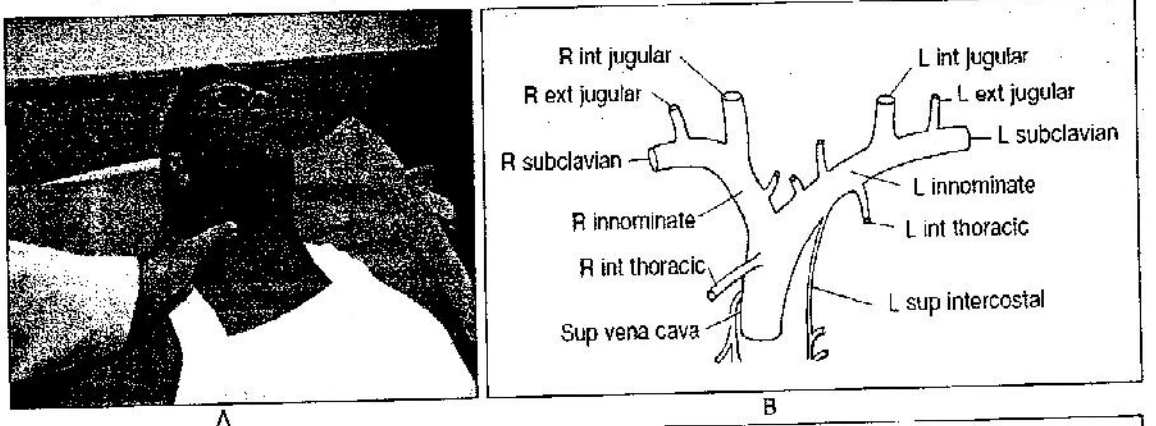


FIGURE 2 – 42. Palpation of the carotid artery (A) and the anatomy of the jugular veins (B).

JUGULAR VENOUS PRESSURE (JVP)

Due to the anatomy of the innominate veins (FIGURE 2 – 42), the JVP is best examined on the patient's right side. The JVP is best estimated from the internal jugular vein, usually on the right side, since the right internal jugular vein has a more direct anatomic channel into the right atrium. The internal jugular veins lie deep to the sternomastoid muscles in the neck and are not directly visible (FIGURE 2 – 43), so the candidate must learn to identify the pulsations of the internal jugular vein that are transmitted to the surface of the neck, making sure to carefully distinguish these venous pulsations from pulsations of the carotid artery (BOX 2 – 9). If pulsations from the internal jugular vein cannot be identified, those of the external jugular vein can be used. The external jugular vein is more superficial, prominent and generally easier to see (FIGURE 2 – 43). Although it can be obstructed as it traverses the deep fascia of the neck, this rarely presents a problem. If it is visible, pulsatile and not obstructed, it can be used to assess JVP.

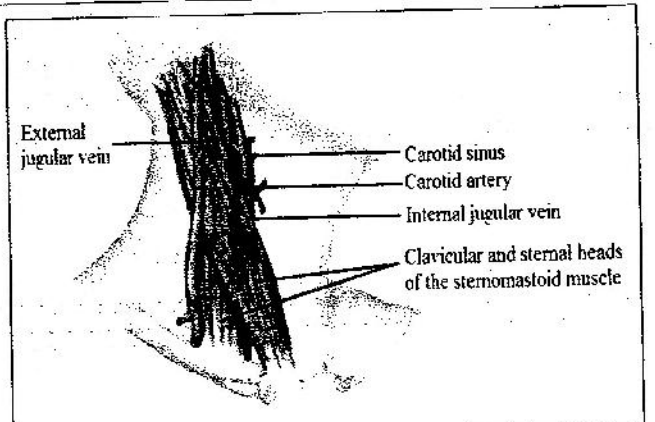


FIGURE 2 – 43. Jugular veins relationships

The sternal angle is approximately 5 cm above the right atrium and the vertical distance between the top of the oscillatory venous column and the level of the sternal angle should extend not more than 4 cm. The JVP is thus < 9 cm H2O (7 mmHg) which reflects the right atrial pressure and indirectly the right ventricular function. When the patient is placed at 45° with the head supported and turned a few degrees to the left, the JVP becomes visible along the line of the sternomastoid muscle. The normal Jugular venous pressure reflects phasic pressure changes in the right atrium and consists of two or sometimes three positive waves and two negative troughs (FIGURE 2 – 43).

BOX 2 – 9. Distinguish the jugular venous pulse from the carotid pulse

	Jugular venous pulse	Carotid pulse
1	It is impalpable (visible only)	Visible and palpable
2	Varies with position and respiration	No variation
3	Most prominent movement usually inward	Most prominent movement usually outward
4	Two peaks per heart beat (in sinus rhythm)	One peak per heart beat
5	Increases with abdominojugular reflux	No effect
6	Obliterated by finger pressure on the neck root	No effect
7	Moves ear lobes when very high	Does not move ear lobes

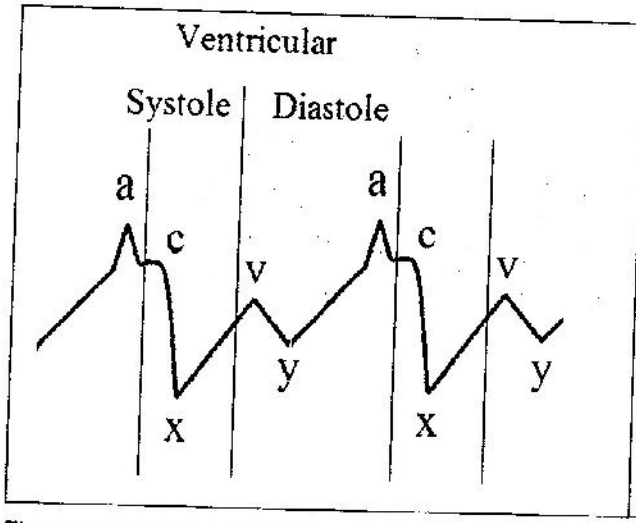


FIGURE 2 – 44. The JVP wave forms. To remember the jugular venous waves look at the following points:

1. The upper waves are c, v, and a mimicking CVA (stroke).
2. The systolic waves are the c and the x waves; all in this field (c, x, and systole) containing a pronunciation of s.

The x descent reflects relaxation and fall in pressure of right atrium. The v wave reflects passive right atrial filling. The y descent reflects passive filling of the right ventricle and the opening of the tricuspid valve. The a wave reflects right atrial contraction. The c wave reflects closure of the tricuspid valve (not usually visible). The two peaks of the a and v wave are the most easily seen. The most common cause of a high venous pressure is an elevated right ventricular diastolic pressure (BOX 2 – 10). In patients suspected of having right ventricular failure who have a normal jugular venous pressure at rest, the abdominojugular reflex may be helpful. The palm of the candidate's hand is placed over the abdomen, and a firm pressure is applied for 10 seconds or more. In normal subjects, this maneuver does not alter the jugular venous pressure significantly, but when right heart function is impaired, the upper level of the venous pulsation usually increased. A positive abdominojugular test is best defined as an increased in jugular venous pressure during 10 seconds of firm mid-abdominal compression followed by a rapid drop in pressure of 4 cm blood on release of the compression.

BOX 2 – 10. Causes of raised jugular venous pressure

Condition	Comment
Physiological elevation	Pregnancy and during expiration
Elevation	Heart failure and fluid overload
Non-pulsatile elevation	Superior vena cava obstruction
Loss of a wave	Atrial fibrillation
Giant (prominent) a wave	Pulmonary stenosis, pulmonary hypertension, pulmonary embolism, and tricuspid stenosis
Giant v wave	Tricuspid regurgitation
Cannon a wave	1. Regular in ventricular tachycardia and supraventricular tachycardia 2. Irregular in complete heart block, right ventricular chamber pacing, and ventricular extrasystole
Prominent y and x descent	Constrictive pericarditis
Prominent y descent and absent x descent	Pericardial tomponade

The examination sequence of jugular venous pressure is conducted as follows:

STEP I Position the patient until the waveform is clearly visible. Start at semirecumbent posture with the head of the bed elevated 45° from the horizon. If the JVP is low, the patient may have to lie flat for it to be seen; if high, the patient may need to sit upright.

STEP II Ensure that the neck muscles are relaxed by resting the back of the head on a pillow

STEP III Look across the neck from the right side of the patient (no way to check the JVP from the left side) to identify the internal jugular vein and the external jugular vein. The internal jugular vein enters the neck behind the mastoid process. It runs deep to the sternocleidomastoid muscle before entering the thorax between the sternal and clavicular heads and can only be examined when the neck muscles are relaxed (FIGURE 2 – 43). Although the vein itself cannot be seen, a diffuse pulsation is visible when the pressure in the internal jugular vein is elevated.

STEP IV If pulsations from the internal jugular vein cannot be identified, those of the external jugular vein can be used. The external jugular vein passes diagonally over the surface of the sternomastoid and is lateral to the internal jugular vein (FIGURE 2 – 43).

STEP V Differentiate the jugular vein pulsation from the carotid pulsation. The jugular venous pulse has two peaks per heart beat (in sinus rhythm) and the most prominent movement is inward.

STEP VI Simultaneously palpation of left carotid artery aids the candidate in deciding which pulsation is venous.

STEP VII Palpate the pulsation in the right side of the neck. The jugular venous pulse is not palpable.

STEP VIII Perform the occlusion test to obliterate the pulsation of the jugular veins at the neck root.

STEP IX Perform the abdominojugular reflux to assess the right heart function.

STEP X Asking the patient to slowly and fully breathe in and out while the candidate is looking at the patient's JVP aids in demonstrating the Kussmaul sign. Normally the JVP falls with inspiration and increases with expiration; the reverse of that indicates positive Kussmaul sign. Kussmaul sign is a paradoxical rise in JVP during inspiration. It occurs in severe right-sided heart failure and constrictive pericarditis but may also occur in tricuspid stenosis, right ventricular infarction, and restrictive cardiomyopathy.

STEP XI The JVP is the vertical height in centimetres between the top of the venous pulsation and the sternal angle, whether they are sitting at 45° or not (FIGURE 2 – 45)

STEP XII Identify the timing and form of the pulsation and note any abnormality.

FIGURE 2 – 45. JVP assessment requires the patient in semi-recumbent posture (A), simultaneous palpation of the left carotid (B); palpation of right neck pulsation (C); obliteration of the neck veins (D), abdominojugular reflex (E), and measurement of JVP (F and G). (H) shows typical example of raised internal jugular venous pressure.

THE CHEST

Full chest assessment should include breast examination, respiratory system examination, and examination of the precordium (heart examination). Breast examination is not part of the general examination and is dealt with separately. Like wise the precordium and the respiratory system are systemic examination. Therefore the upper chest is examined during the general examination. It is advisable to look for the following chest abnormalities:

Patient with jaundice	The chest is inspected for spider naevi and gynecomastia. Spider naevi (spider angiomas) are isolated telangiectases that characteristically fill from a central feeding vessel found in the distribution of the superior vena cava on the upper trunk, arms and face (FIGURE 2 – 46). Women may have up to five spider naevi in health; palmar erythema and numerous spider naevi are normal during pregnancy. Gynaecomastia which means breast enlargement in men (FIGURE 2 – 46).
Patient with suspected SVC obstruction	The upper chest is inspected for edema, cyanosis, and telangiectasia (FIGURE 2 – 46).
Patients with uremia	Look for double lumen catheter
Patients with cardiac disease	Look for pacemaker or implantable cardioverter defibrillator

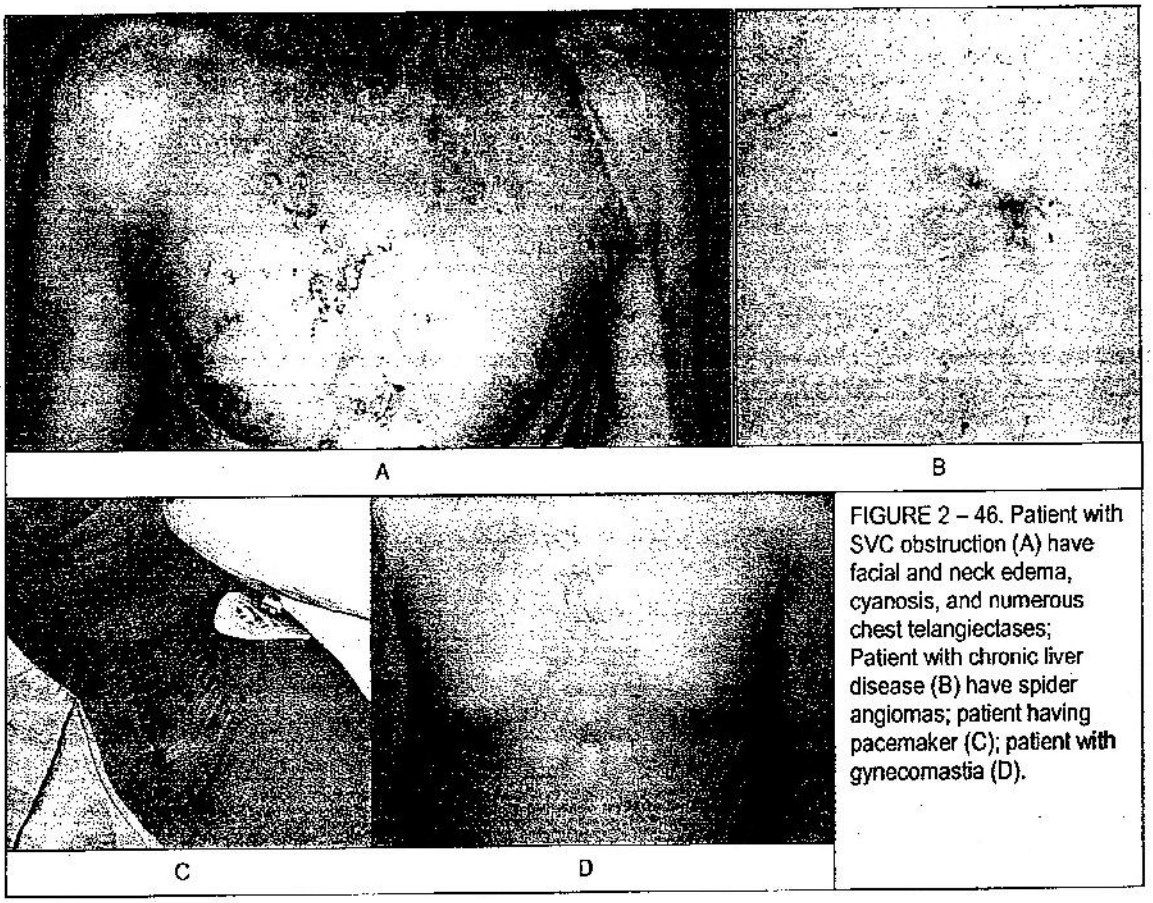


FIGURE 2 – 46. Patient with SVC obstruction (A) have facial and neck edema, cyanosis, and numerous chest telangiectases; Patient with chronic liver disease (B) have spider angiomas; patient having pacemaker (C); patient with gynecomastia (D).

THE ABDOMEN

The abdomen examination is part of the systemic examination and is not usually examined during general examination.

THE LOWER LIMBS

Like that of the hands, examination of the lower limbs (FIGURE 2 – 47) includes the following steps:

STEP I Inspect the outer and inner borders of the legs as well as the dorsal and the planter surfaces of the feet for posture, shape, size, and color.

STEP II Examine for toe nails abnormalities which are as the same as that found in hand nails.

STEP III Feel the pulses of the dorsalis pedis, anterior and posterior tibial arteries

STEP VI Feel the temperature of both legs. Use the dorsal surface of your finger to feel the temperature over the dorsal surface of the legs (Rule: back over back)

STEP V Examine for edema

In hemiplegia (one side paralysis), the upper arms are held tight to the sides with elbows, wrists, and fingers flexed. The legs are extended and internally rotated; the feet are plantar flexed. Foot drop, due to common peroneal nerve palsy, is characterised by weakness of foot dorsiflexion and eversion.
Deviation of the part of the body away from the midline is called valgus deformity and towards the midline, varus deformity. Pes planus (flat feet) or pes cavus (high-arched foot) may also be seen. In hallux valgus, the great toe is abnormally abducted in relationship to the first metatarsal, which itself is deviated medially. A bunion (a soft tissue bursal swelling) may develop over the protuberant first metatarsal head and become inflamed or infected. As the condition progresses, the hallux may rotate over the second toe. Claw toes result from dorsiflexion (hyperextension) at metatarsophalangeal (MTP) joints and plantar flexion at proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints. Hammer toes are due to dorsiflexion at MTP and DIP joints and plantar flexion at PIP joints. Mallet toes describe plantar flexion at DIP joints. Signs of arthritis include swollen red tender toe joints that occur in rheumatoid arthritis, psoriasis, or gout. The first attack of gout most commonly affects the first metatarsophalangeal joint; the attack is termed podagra. With persistent inflammation, rheumatoid arthritis may result in joint changes in the feet, including eversion, hallux valgus and lateral deviation and dorsal subluxation of the toes. Osteoarthritis of the toes also produces nodular changes of Bouchard and Heberden's nodules. Osteoarthritis of the knee joint often produces swelling and distortion of the surface markings of the joint. With advanced damage the joint becomes unstable and may develop a varus or a valgus deformity.
The feet are enlarged, broad and bulky in acromegaly. It is caused by thickening of the skin and soft tissues, and is particularly noticeable over the heels and the lateral borders. Sometimes the pads of the feet are so thickened that the toes cannot be placed on the floor. In pseudohypoparathyroidism, the short third, fourth and fifth metatarsals create dimples on the dorsum of the corresponding foot, with shortening of one or more toes. Polydactyly is characteristically seen in certain congenital syndrome e.g. Lawrence-Moon-Biedl syndrome. Marfan's syndrome is also notified by looking at the feet for the presence of arachnodactyly.
Xanthomas, deposits of fatty material into the skin, subcutaneous fat and tendons, may be the first clue to primary or secondary hyperlipidemia. Various clinical patterns are seen which correlate well with the underlying cause:

1	Tuberous xanthoma	Firm yellow nodules and plaques on extensor surfaces	Due to hypercholesterolemia
2	Tendon xanthoma	Firm subcutaneous swellings attached to tendons	Due to hypercholesterolemia
3	Palmer xanthoma	Yellow macules often affecting palmar creases	Due to hypercholesterolemia
4	Eruptive xanthoma	Numerous small yellowish papules commonly affecting the extensor surfaces e.g. elbow or knee	Due to hypertriglyceridemia

Peripheral arterial insufficiency produces profound changes in the appearance of the legs. Often there is pallor of the legs, cyanosis, coldness, hair loss, and muscular wasting with small areas of infarction, ulceration, and gangrene. Unlike neuropathic ulcers associated for example with diabetes which are often painless, ischemic ulcers usually cause severe pain, especially on elevation of the leg (BOX 2 – 11)

BOX 2 – 11. Sites of various types of ulcer

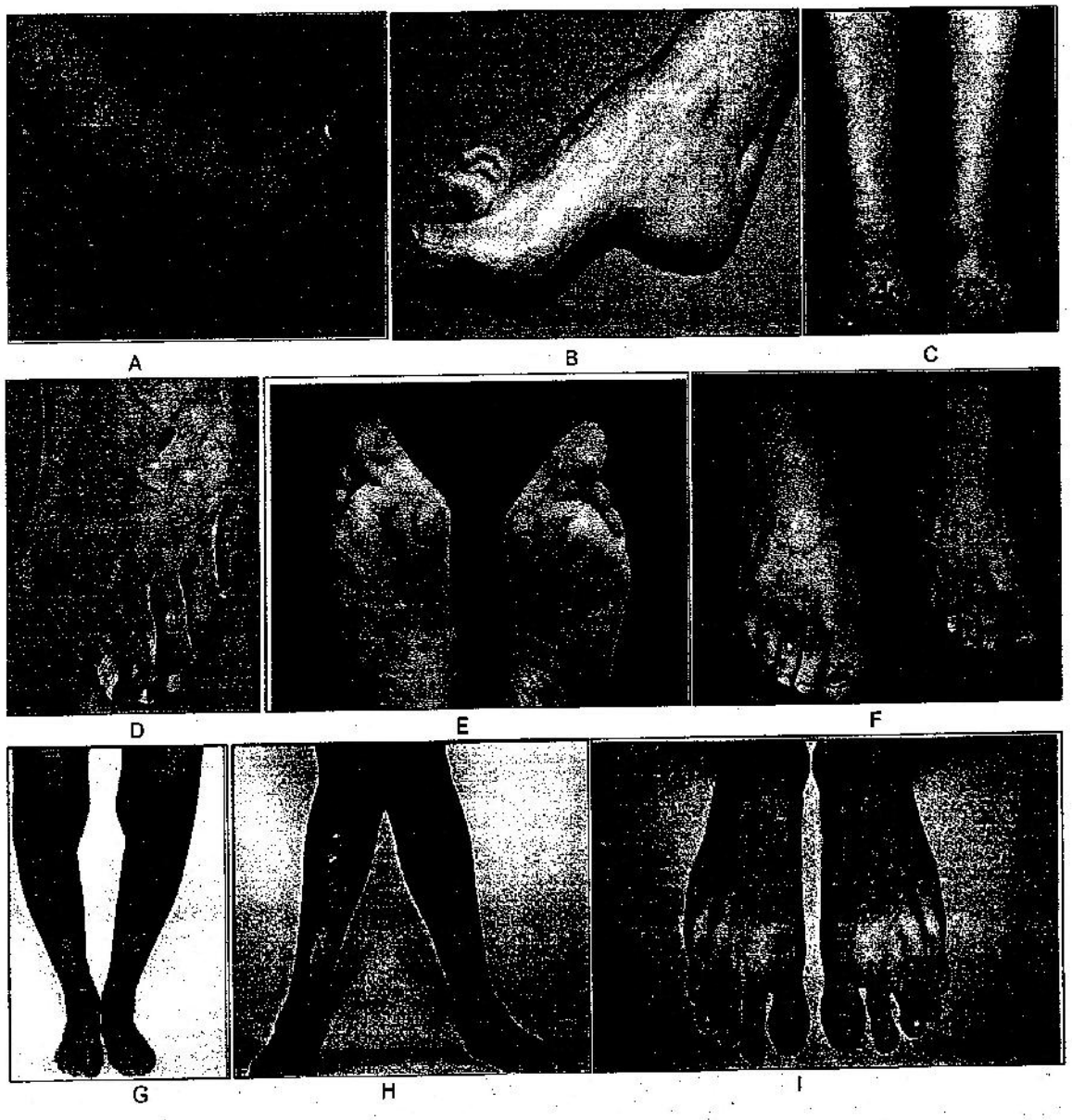
Ischemic ulcer	Venous ulcer	Neuropathic ulcer
It is found in the pressure areas e.g. malleoli, heel, fifth metatarsal base, metatarsal heads and toes	It is found in the inner and some times the outer ankle	It is found in pressure points in areas with diminished sensation e.g. the heel, ball of the foot or toes.

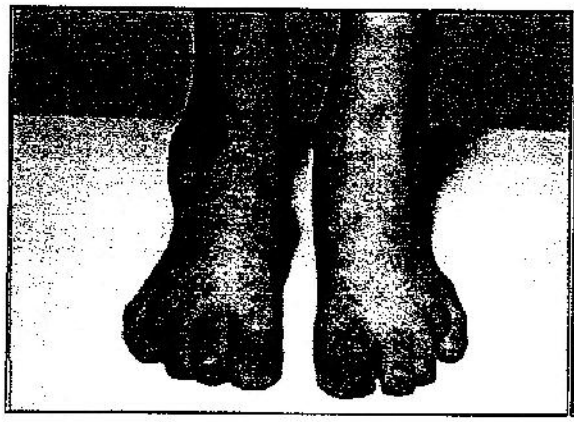
In livedo reticularis, localized areas of the extremities develop a mottled or net – like appearance of reddish to blue discoloration. The primary form of this disorder is benign, occurs more frequently in women than in men, and the most common age of onset is in the third decade. Secondary livedo reticularis can occur with atheroembolism, SLE anticardiolipin antibodies, vasculitides, hyperviscosity, and in patients receiving large doses of vasopressors e.g., epinephrine, norepinephrine, and dopamine.

Buerger's disease produces ulceration and gangrene most frequently in the distal half of the digits around the margins of the nails. Venous problems in the form of deep venous thrombosis (DVT) or varicose veins may also be noted.
Planter thickening occurs in eczema, familial Tylosis syndrome, and in Keratoderma blennorrhagicum.
Persistent pressure that is greater than capillary pressure which lasts for 2 hours can cause ischemia and resultant ulceration (bed sores). Therefore, immobilized patients should be turned at least every 2 hours. The most common sites of pressure ulcers are the sacrum, thighs, heels, and lateral malleoli. Bed (pressure) sores are usually graded into four grades:

Grade I	Erythema, skin intact
Grade II	Skin loss, epidermis or dermis (abrasion, blister, shallow crater)
Grade III	Full thickness loss and damage to subcutaneous tissues
Grade IV	Extensive destruction, tissue necrosis or damage to underlying muscle or bone

Vasculitic skin rashes may include palpable purpura, livedo reticularis, subcutaneous nodules, ulcers, pulp infarction and digital gangrene.





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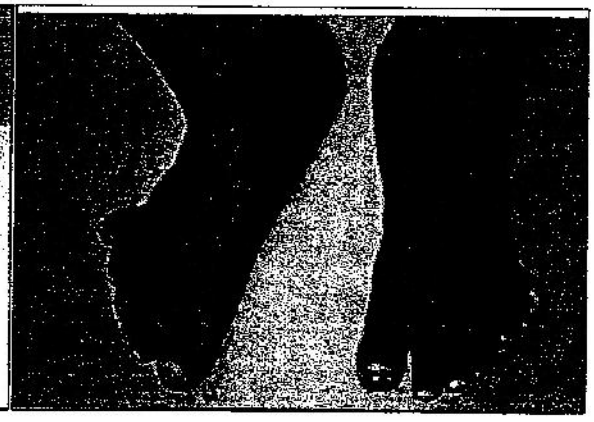

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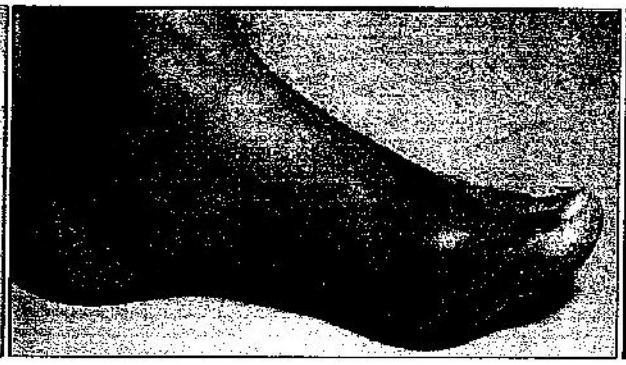

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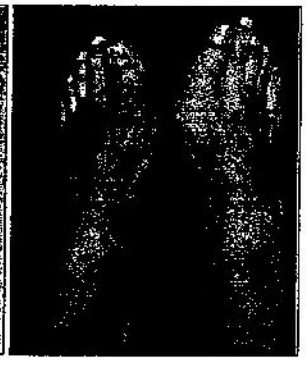

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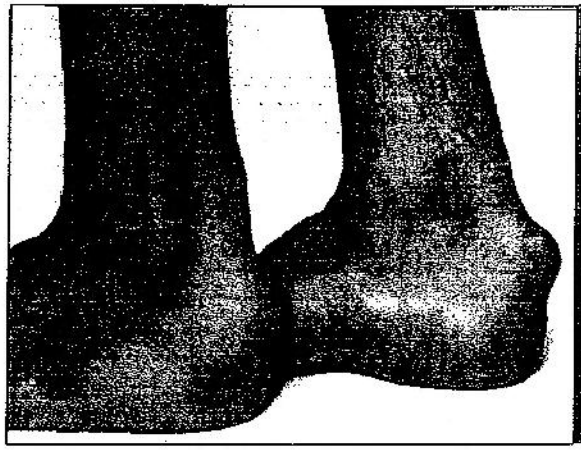

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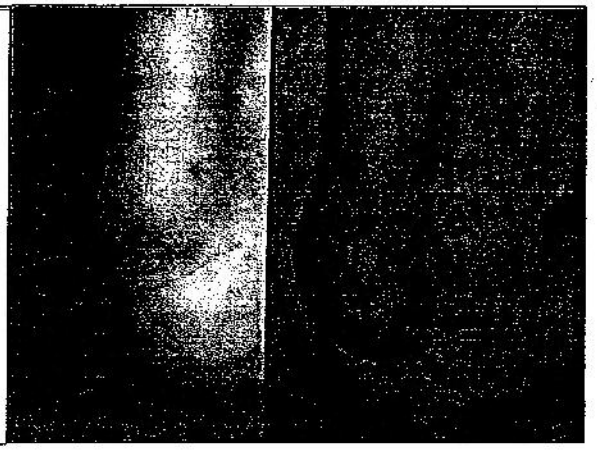

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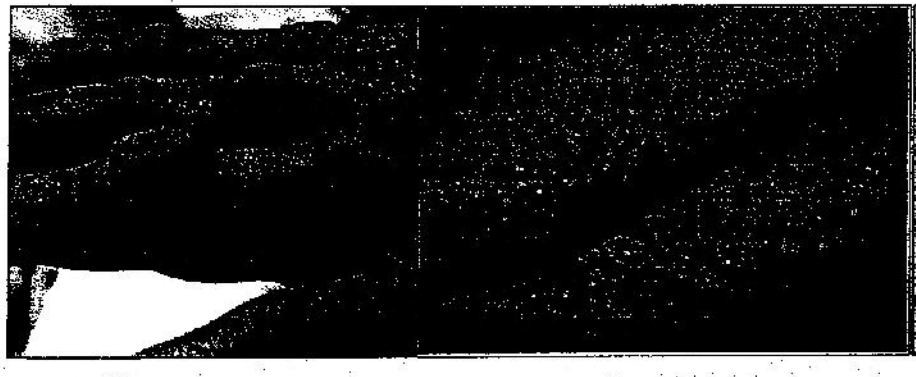

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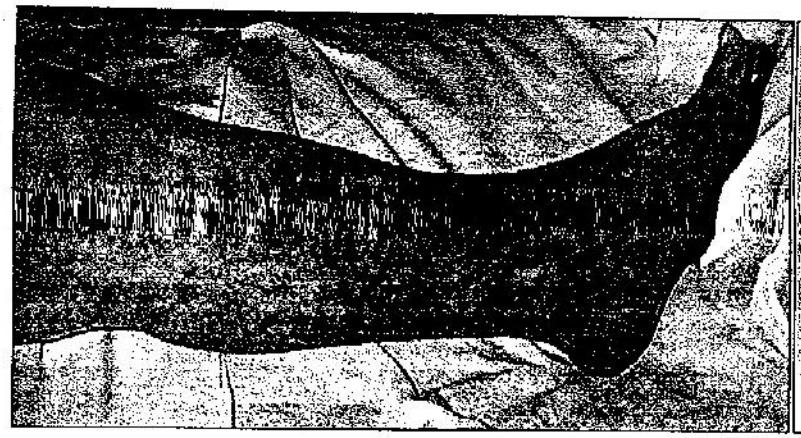

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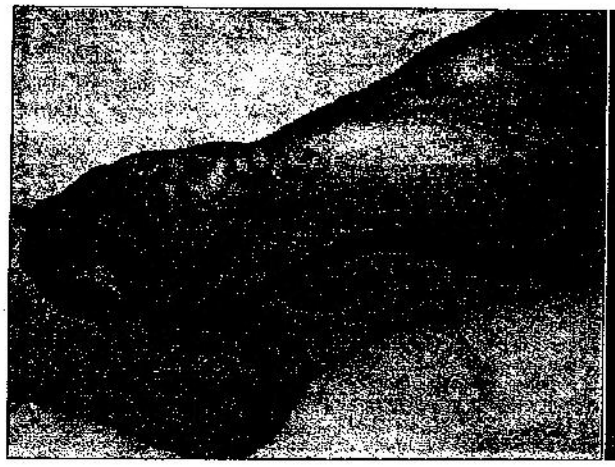

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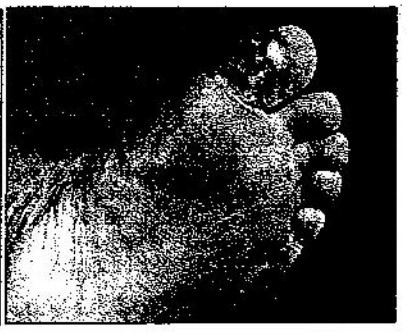

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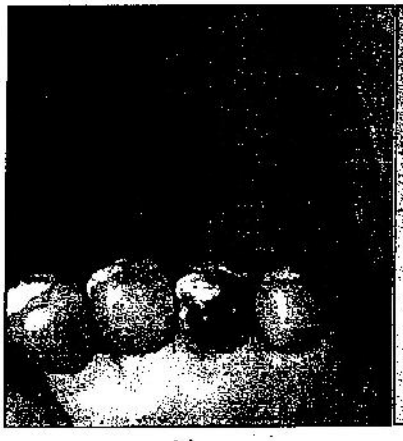

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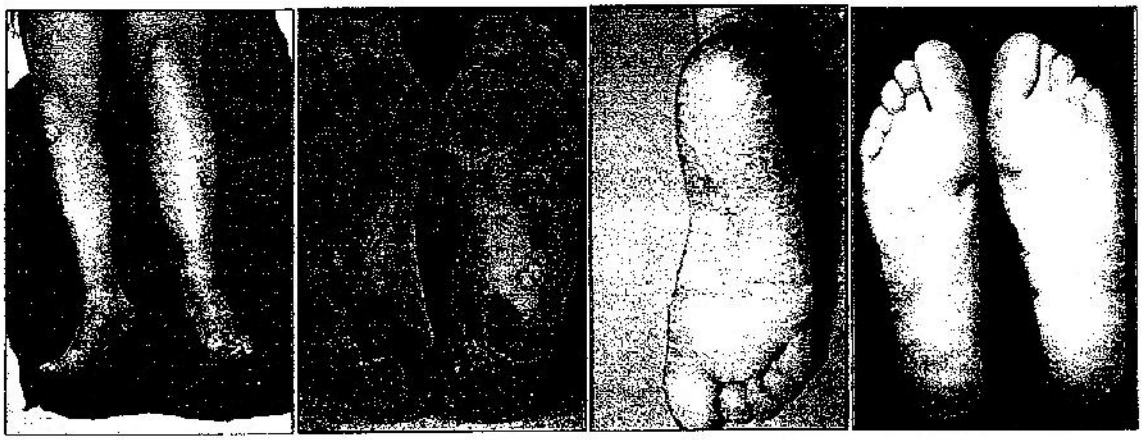
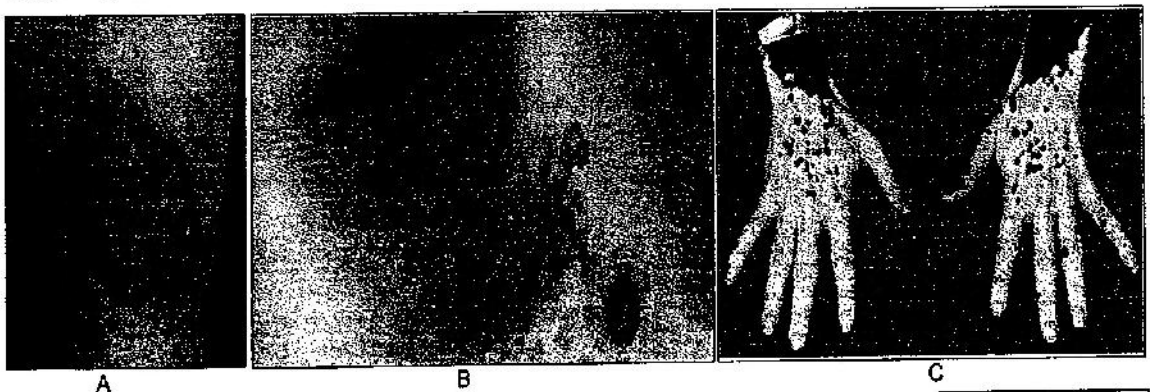


DD EE FF GG

FIGURE 2 – 47. Various clinical findings in the lower limbs including pes cavus and hammer toe (A), pes cavus and claw toes (B), claw toe (C), hallux valgus (D), hallux valgus with callosities at the metatarsal heads (E), large foot of acromegaly as compared with normal foot (F), valgus deformity of the knee (genu valgus) (G), varus deformity of the knee (genu varus) (H), arachnodactyly of Marfan's syndrome (I), short fourth metatarsal bones bilaterally due to pseudohypoparathyroidism (J), polydactyly (K), rheumatoid nodule at the Achilles tendon (L), left podagra in lateral (M), and anteroposterior views (N), tendon xanthomas of the Achilles tendon (O) and (P), tuberous xanthoma (Q), eruptive xanthoma (R), ischemia of the limb with dusky colored foot (S), ischemia of the whole left leg with bluish discoloration and erythema (T), dry gangrene (U), livedo reticularis (V), gangrene in Buerger's disease (W), ischemic (arterial) ulcer (X), venous ulcer (Y), neuropathic ulcer (Z), vasculitis manifested as dry gangrene (AA), pressure (bed) sores (BB), varicose veins (CC), deep venous thrombosis (DD), Keratoderma blennorrhagicum (EE), Tylosis of the planter surface (FF), and carotenoderma (GG).

Endocrine leg signs (FIGURE 2 – 48) may include diabetic skin complication and pretibial myxoedema of hyperthyroidism. The most common skin manifestation of diabetes mellitus is skin infection with the resultant protracted wound healing and skin ulcerations. Diabetic dermopathy or shin spots begin as an erythematous area and evolve into an area of circular hyperpigmentation. These lesions result from minor mechanical trauma in the pretibial region. Necrobiosis lipoidica diabeticorum usually begins in the pretibial region as an erythematous plaque or papules that gradually enlarge, darken, and develop irregular margins, with atrophic centers and central ulceration. They may be painful. Vitiligo occurs at increased frequency in type 1 diabetes. Acanthosis nigricans (hyperpigmented velvety plaques seen on the neck, axilla, or extensor surfaces) is sometimes a feature of severe insulin resistance and accompanying diabetes. Granuloma annulare (erythematous plaques on the extremities or trunk) and scleredema (areas of skin thickening on the back or neck at the site of previous superficial infections) are more common in the diabetic population. Lipoatrophy and lipohypertrophy can occur at insulin injection sites but are unusual with the use of human insulin.

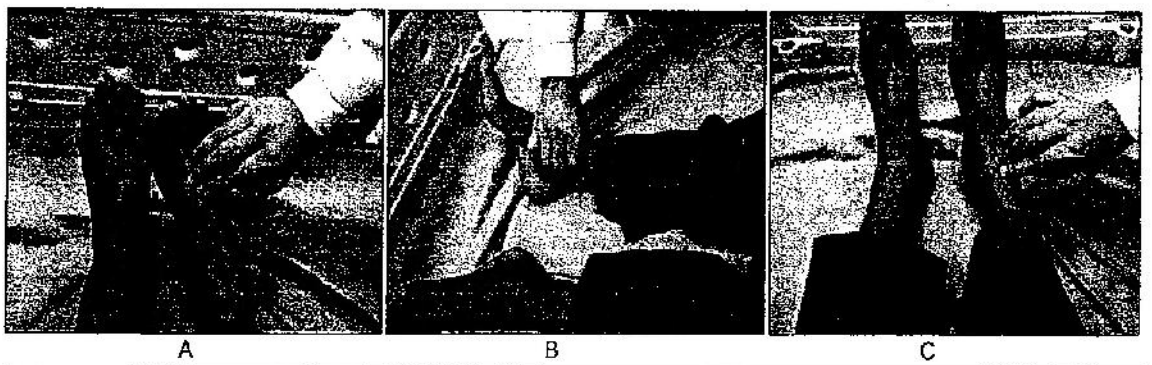


A B C

FIGURE 2 – 48. Some clinical skin findings in patients with diabetes mellitus including necrobiosis lipoidica diabeticorum (A), acanthosis nigricans (B), and vitiligo (C).

Dorsalis pedis artery (FIGURE 2 – 49) can be felt when the candidate presses the tips of the fingers upon cleft between the first and second metatarsal bones (just lateral to the tendon of extensor hallucis longus). Patient's left dorsalis pedis artery examined more easily than the right.

Posterior tibial artery can be felt in the midway between medial malleolus and the medial border of Achilles tendon as shown in FIGURE 2 – 49. Anterior tibial artery (FIGURE 2 – 49) is felt anteriorly mid-way between the lateral and the medial malleoli. The candidate duty is determine just whether these vessels are patent or not.



A B C

FIGURE 2 – 49. The clinical techniques used to determine the lower limbs vessels including dorsalis pedis artery (A), posterior tibial artery (B), and anterior tibial artery (C).

EXAMINATION OF LEG EDEMA

Tissue spaces are divided into intracellular spaces and extracellular spaces. Extracellular space is composed of intravascular and interstitial compartments. Edema is defined as a collection of fluid in the interstitial tissue space.

In the examination of edema the candidate should determine whether the edema is unilateral or bilateral, symmetrical or asymmetrical, pitting (FIGURE 2 – 50) or non-pitting, tender or not, and its grade and level. Examination of leg edema (FIGURE 2 – 51) is conducted in the following way:

1 STEP I The patient must lie flat quite comfortably

2 STEP II Inspect the legs and ankle for swelling

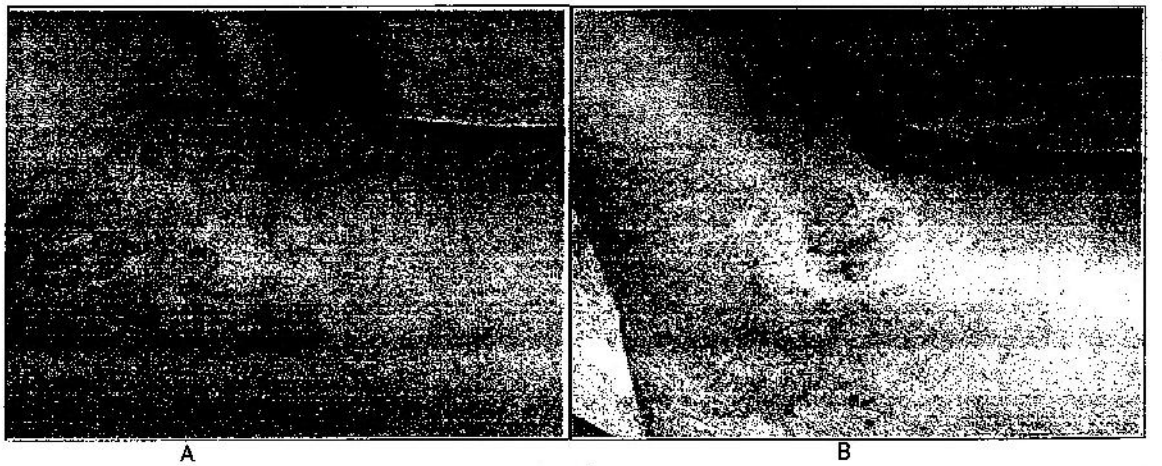
3 STEP III Use your thumbs and simultaneously apply firm pressure over a bony prominence e.g. shin or lateral malleoli to determine whether the edema is pitting or not.

4 STEP IV The pressure is maintained for few seconds while you watch the face of the patient to determine whether the edema is tender or not

5 STEP V Palpate the area that you just pressed to see the indentation (pit) made by your pressure and indicates that the edema is pitting. Determine whether the edema is symmetrical or not. Pitting edema is usually quantified from 1 + to 4 + depending on how long the indentation persists (from slight 1 + to very marked 4 +)

6 STEP VI If the leg is grossly swollen, press at a higher level to establish how far edema extends (level of edema)

In ambulant patients, the edema first appears at the ankles and over the dorsum of the foot. In bed-bound patients, edema often appears first over the sacrum. Causes of edema are shown in BOX 2 – 12.



A B

FIGURE 2 – 50. Pitting leg edema

FIGURE 2 – 51. Examination of leg edema including inspecting the feet, ankles, and leg (A) for swelling, applying light pressure (B) for few seconds whilst you look at the patient's face to determine whether the edema is tender or not, and then feel the pressed points to determine the edema is pitting or not.

BOX 2 – 19. Causes of edema

Unilateral leg edema

1. Cellulites
2. Deep venous thrombosis (DVT)
3. Lymphoedema:
   - Congenital (Milroy disease).
   - Acquired (postinfectious, obstructive as a result of pelvic neoplasm, or iatrogenic after surgical removal of lymph nodes)
4. Calf hematoma
5. Ruptured baker's cyst

Bilateral

Non pitting edema
- Myxoedema
- Chronic lymphoedema

Pitting edema
1. Cardiac causes
   - Congestive heart failure
   - Right sided heart failure
   - Cardiac tomponade
   - Constrictive pericarditis
2. Renal causes
   - Renal failure
   - Nephrotic syndrome
   - Nephritic syndrome
3. Liver causes
   - Liver failure
   - Liver cirrhosis
4. Protein – losing enteropathy

In contrast to edema, myxoedema is characterized by swelling which does not pit on pressure and is due to infiltration of the tissue by a firm mucinous material. Edema must be distinguished from lymphoedema (FIGURE 2 – 52). Venous disease produces pitting edema and an increased superficial venous pattern, involves the foot but not the toes, and is associated with relatively normal skin. Lymphoedema on the other hand is nonpitting, does not result in superficial venous prominence, involves the foot and the toes, and is associated with thickened skin.

FIGURE 2 – 52. Some causes of unilateral leg swelling including ruptured baker's cyst (A), lymphoedema (B), and deep venous thrombosis (C).

PRIMARY AND SECONDARY SKIN LESIONS

Undergraduate third and fourth medical students may face a difficulty understanding such lesions; they are, of course, in need for assistance from their older colleagues. All candidates should know primary and secondary skin diseases. Some of the primary skin lesions are described here and shown in FIGURE 2 – 53. Macule is a flat, colored lesion, < 0.5 cm in diameter, not raised above the surface of the surrounding skin. Patch is a large (> 0.5 cm) flat lesion with a color different from the surrounding skin. This differs from a macule only in size. Nonpalpable purpura is a flat lesion that is due to bleeding into the skin; if <3 mm in diameter, the purpuric lesions are termed petechiae; if >3 mm, they are termed ecchymoses. Palpable purpura is a raised lesion that is due to inflammation of the vessel wall (vasculitis) with subsequent hemorrhage. Papule is a small, solid lesion, <0.5 cm in diameter, raised above the surface of the surrounding skin and hence palpable. Nodule is a larger (0.5–5.0 cm), firm lesion raised above the surface of the surrounding skin. This differs from a papule only in size. Plaque is a large (>1 cm), flat-topped, raised lesion; edges may either be distinct or gradually blend with surrounding skin. Vesicle is a small, fluid-filled papule, <0.5 cm in diameter, raised above the plane of surrounding skin. Pustule is a vesicle filled with pus. Bulla is a fluid-filled, raised, often translucent lesion >0.5 cm in diameter. The bulla may be tense or flaccid and may be on a base of normal or erythematous skin. Wheal is a raised, erythematous, edematous papule or plaque, usually representing short-lived vasodilatation and vasopermeability. Telangiectasia is a dilated, superficial blood vessel. Burrow is a linear or curvilinear papule, caused by a burrowing scabies mite.

CLINICAL CLUE

When the platelet count is markedly decreased, petechiae first appear in areas of increased venous pressure, ankles and feet in an ambulatory patient. Petechiae are usually a sign of a decreased platelet number and not platelet dysfunction (Rule: low Platelets = Petechiae). Wet purpura, blood blisters that form on the oral mucosa, are thought to denote an increased risk of life-threatening hemorrhage in the thrombocytopenic patient (Rule: Wet purpura = Warning sign). Excessive bruising is seen in disorders of both platelet number and function.

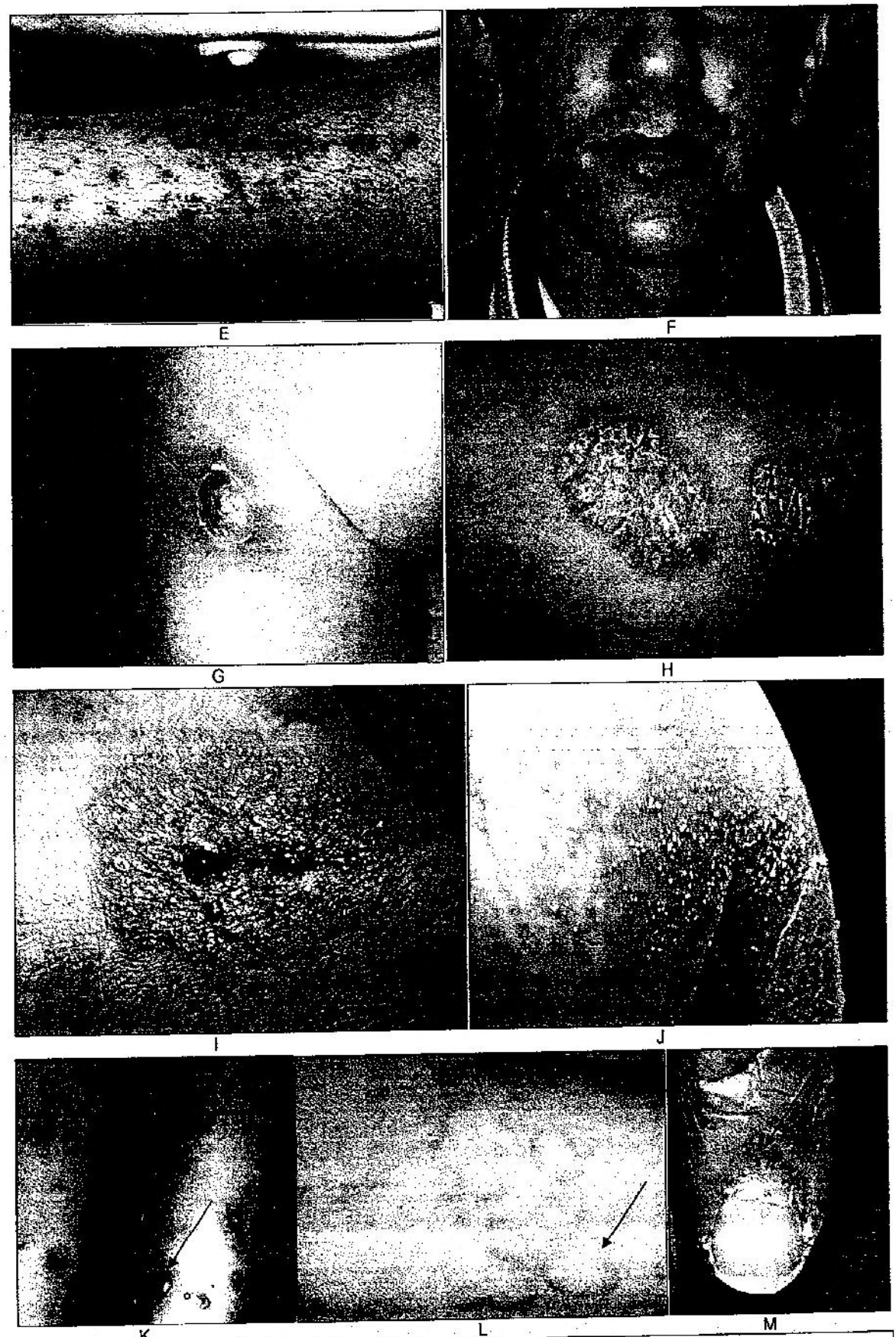


FIGURE 2–53. Primary skin lesions including macule (A), patch (B), petechiae (C), ecchymoses (D), palpable purpura (E), papule (F), nodule (G), plaque (H), vesicle (I), pustule (J), bulla (K), wheal (L), and telangiectasia (M).

Distinguishing primary from secondary lesions is one of the most important parts of the skin examination. Secondary lesions (FIGURE 2 – 54) are modifications, usually made by the patient, of the primary lesion. Lichenification is a distinctive thickening of the skin that is characterized by accentuated skin-fold markings. Scale is an excessive accumulation of stratum corneum. Crust is a dried exudate of body fluids that may be either yellow (i.e., serous crust) or red (i.e., hemorrhagic crust). Verruca is a lesion with papillomatous or warty changes on the surface. Erosion means loss of epidermis without an associated loss of dermis. Ulcer means loss of epidermis and at least a portion of the underlying dermis. Excoriation is linear, angular erosions that may be covered by crust and are caused by scratching. Atrophy is an acquired loss of substance. In the skin, this may appear as a depression with intact epidermis (i.e., loss of dermal or subcutaneous tissue) or as sites of shiny, delicate, wrinkled lesions (i.e., epidermal atrophy). Scar is a change in the skin secondary to trauma or inflammation. Sites may be erythematous, hypopigmented, or hyperpigmented depending on their age or character. Sites on hair-bearing areas may be characterized by destruction of hair follicles.

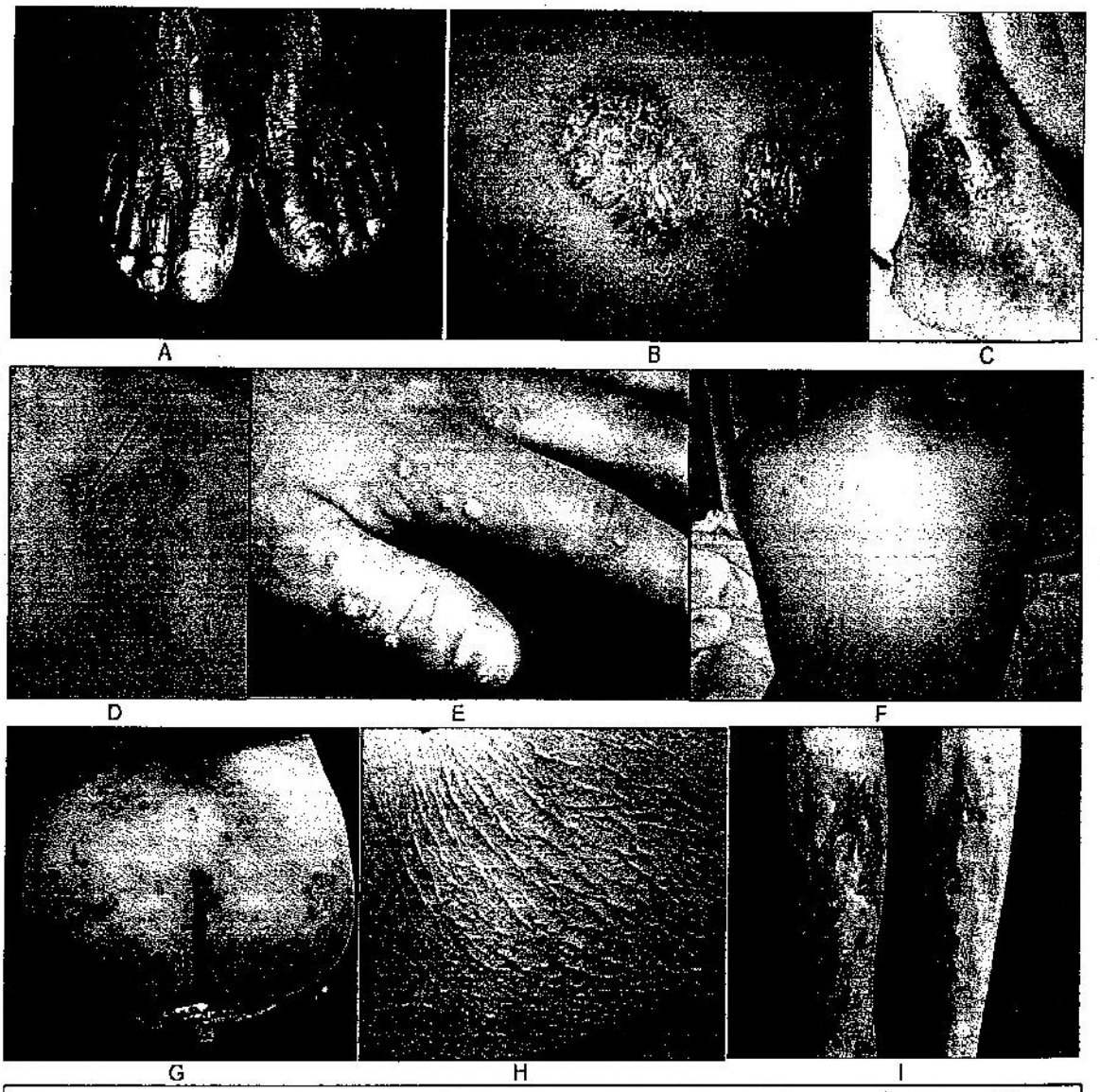


FIGURE 2 – 54. Secondary skin lesions including lichenification (A), scale (B), ulcer (C), crust (D), Verruca (E), excoriation (F), erosion (G), atrophy (H), and scar.

MEDICAL ACCESSORIES

The candidates should know how and when to use the following medical equipments (FIGURE 2 – 55):

1 Intravenous cannulae
2 Oxygen mask
3 Nasal prongs
4 Folly's catheter
5 Blood unit
6 Fluid unit
7 Draining bag
8 Surgical dressing
9 Naso-gastric (NG) tube
10 Chest tube
11 Colostomy bag
12 Radivac drain
13 External fixation
14 Inhalers and nebulizers
15 Cardiac monitors
16 Direct current (DC) shock devices

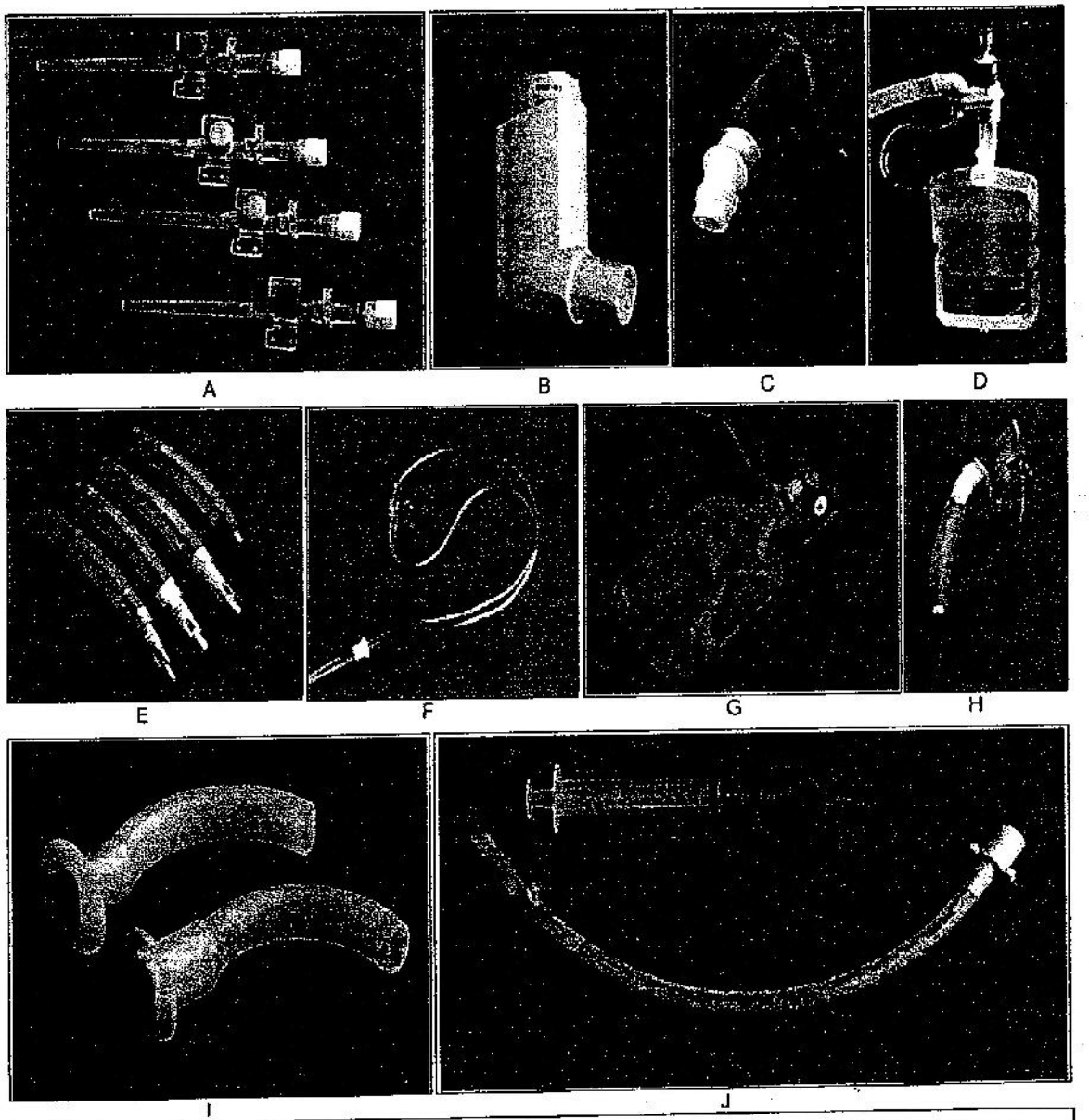


FIGURE 2 – 55. Medical accessories including intravenous cannulae (A), metered dose inhaler device (B), low flow/variable oxygen concentration mask (C), humidification circuit to humidify the incoming oxygen (D), Venturi valves (E), nasal cannulae (prongs) (F), non re-breather mask (G), mask with Venturi valve attached (H), oropharyngeal airways (I), and endotracheal tube (J).

LEARNING POINTS IN THE PRESENTATION OF GENERAL EXAMINATION

1 The candidate should train him/her self well during clinical course aiming to complete the general physical examination in 3 – 5 minutes
2 The examination should always be considered from the right side of the patient even when the examination committee stands at that side
3 It is essential to take permission from the patient before starting the examination saying for example: Good morning (or how are you?), I m Dr. Ahmed, a third year medical student. Can I examine you?
4 Ensure complete privacy to the patient by pulling the curtains around his/her bed
5 The candidate should get first impression that include Age group of the patient, Sex, Awareness, Decubitus of the patient in the bed, state of Comfortability, Body built, Dyspnea and tachypnea. These physical signs should always be presented at the start of the presentation. A useful mnemonic is A SAD CBD. He/she should then examine the patient thoroughly taking particular consideration to Jaundice, Anemia, Cyanosis, Clubbing, Oedema, and Lymphadenopathy. These signs are collectively known as JACCOL.
6 The upper part of the chest should be part of the general examination particularly when the clinical setting is relevant e.g. SVC obstruction
7 In long case examination the presentation should include the history, the general examination, vital signs, examination of the involved system, and finally the remaining uninvolved systems. An example for that is when a patient presented with melena; the presentation should therefore include the history, the general examination, vital signs, examination of the abdomen, and finally the examination of the other systems
8 In short case examination the candidate should follow the instruction provided by the examination committee. The same rules are applied to the OSCE examinations
9 The candidate should also be ready for non classic types of examination. Examples for that are:
   Examine the hands of this patient
   Examine the eye of this patient none neurologically
   Examine the neck of this patient

EXAMPLES

Middle aged female lethargic patient sitting uncomfortable in her bed, of thin body built, neither dyspneic nor tachypneic. She had digital clubbing grade three with bilateral flapping tremor and palmer erythema; both radial and brachial arteries were patent with 60 beats per minute and regular rhythm. She had yellowish discoloration of the sclera, pale conjunctiva, no cyanosis with bad oral hygiene. There was no JVP distension, centrally located trachea, no goiter, or lymphadenopathy. Spider angiomas, two in number were present at the upper chest. There was obvious abdominal swelling. There was bilateral symmetrical non-tender pitting grade one leg edema; both dorsalis pedis and posterior tibial arteries were patent. An intravenous cannula inserted into her left arm with three ecchymotic spots beside it.

What is the provisional diagnosis?
The provisional diagnosis is liver failure (hepatic encephalopathy)

Old aged male lethargic patient sitting uncomfortable in his bed, obese, severely dyspneic and tachypneic using accessory muscles of respiration. He had half and half nails, palmer erythema, coarse and bilateral flapping tremor, and multiple ecchymotic spots distributed over both arms. Both radial and brachial arteries were patent with 95 beats per minutes, of good volume and regular. His face was puffy and earthy, pale conjunctivae, corneal arcus, no jaundice, no cyanosis, dry mouth with bad oral hygiene and uremic fetor. JVP was distended to 6 cm above the sternal angle, the trachea was centrally located, and there was no goiter or lymphadenopathy. Double lumen catheter was inserted into the left of his upper chest. There was grade 3 bilateral symmetrical non-tender pitting leg edema. Both dorsalis pedis and posterior tibial arteries were patent. An intravenous cannula was inserted into his right arm as well as a folly's catheter collecting deep color urine.

What is the provisional diagnosis?
The provisional diagnosis is uremia

Old aged male lethargic patient sitting uncomfortable in his bed, obese, severely dyspneic and tachypneic using accessory muscles of respiration. He had palmer erythema, bilateral flapping tremor, and multiple ecchymotic spots distributed over both arms with no digital clubbing. Both radial and brachial arteries were patent; the radial pulse was 95 beats per minutes, of variable volume and irregular rhythm. His face was congested, no pallor, corneal arcus, Xanthelasma over his left lower eye lid, yellowish discoloration of the skin and sclera, central cyanosis, and bad oral hygiene with many missed teeth. His JVP was elevated 11 cm with visible V wave, the trachea was centrally located, and there was no goiter or lymphadenopathy. There was grade was two bilateral symmetrical non-tender pitting leg edema. Both dorsalis pedis and posterior tibial arteries were difficult to be felt. An intravenous cannula was inserted into his left arm as well as a folly's catheter collecting faint colored urine.

What is the provisional diagnosis?
The provisional diagnosis is heart failure with pulmonary edema

Young aged female conscious patient lying comfortable in dorsal decubitus state in her bed, of a thin body built, neither dyspneic nor tachypneic. She had pale palmer creases, no digital clubbing, and no tremor. Both radial and brachial arteries were patent and equal with 100 beats per minutes, of large volume and regular rhythm. Her face was pale, pale conjunctivae, no jaundice, no cyanosis, with bad oral hygiene and three candidal spots were located over her tongue. JVP was not distended, the trachea was centrally located, and there was no goiter. There were three discrete symmetrical rubbery non tender mobile superficial cervical lymph nodes; each was measured three by three cm^2; two of them were on the right and one on the left; no axillary, epitrochlear, or other cervical region lymphadenopathy. There is grade one bilateral symmetrical non-tender pitting leg edema. Both dorsalis pedis and posterior tibial arteries were patent. An intravenous cannula was inserted into her right arm. Multiple petechial rashes were distributed all over her body but particularly around her ankle joints bilaterally.

What is the provisional diagnosis?
The provisional diagnosis is hematological disease e.g. leukemia or lymphoma

Middle aged female conscious patient was lying comfortable in dorsal decubitus state in her bed, obese, but neither dyspneic nor tachypneic. She had pale palmer creases, palmer erythema, no digital clubbing, but nail pitting, and coarse tremor. Hot, red, tender and swollen metacarpophalangeal joints and proximal interphalangeal joints were seen symmetrically and bilaterally. She had swan neck deformity of the left little finger; button-hole deformity of the left index finger; radial deviation of the wrist and ulnar deviation of the fingers. Three subcutaneous nodules were located over the extensor of both arms. Both radial and brachial arteries were patent with 95 beats per minutes, of good volume and regular rhythm. Her face was pale with red eyes, no jaundice, no cyanosis, bad oral hygiene with candidal rash over her left side of the tongue; bad breath smell. JVP was not distended, the trachea was centrally located, and there was no goiter or lymphadenopathy. There was no leg edema. Both dorsalis pedis and posterior tibial arteries were patent. A swelling measured 6 by 6 cm^2 was located in the left popliteal fossa. An intravenous cannula was inserted into his right arm.

What is the provisional diagnosis?
The provisional diagnosis is rheumatoid arthritis

Old aged male lethargic patient sitting uncomfortable in his bed, thin, severely dyspneic and tachypneic using accessory muscles of respiration with pursed-lip breathing. He had warm hands, palmer erythema, bilateral flapping tremor, and multiple ecchymotic spots distributed over both arms with no digital clubbing. Both radial and brachial arteries were patent; the radial pulse was 95 beats per minutes, of large volume, and regular rhythm. His face was dusky, no pallor, corneal arcus, xanthelasma over his left lower eye lid, no jaundice, central cyanosis, and bad oral hygiene with many missed teeth. His JVP was elevated 11 cm with visible a and v waves, the trachea was centrally located, and there was no goiter or lymphadenopathy. There was grade two bilateral symmetrical non-tender pitting leg edema. Both dorsalis pedis and posterior tibial arteries were difficult to be felt. An intravenous cannula was inserted into his left arm as well as a folly's catheter collecting faint colored urine.

What is the provisional diagnosis?
The provisional diagnosis is acute exacerbation of COPD

Young aged female lethargic patient sitting uncomfortable in her bed, thin, severely dyspneic and tachypneic using accessory muscles of respiration with many audible wheezes. She had coarse tremor, thin skin, and multiple ecchymotic spots distributed over both arms with no digital clubbing. Both radial and brachial arteries were patent; the radial pulse was 100 beats per minutes, of large volume, and regular rhythm. She had central cyanosis, no pallor, and no jaundice. She had bad oral hygiene with bad breath smell. Her JVP was not distended, the trachea was centrally located, and there was no goiter or lymphadenopathy. There was no leg edema, and both dorsalis pedis and posterior tibial arteries were patent. She was supplied with nebulizer connecting into an oxygen face mask; an intravenous cannula was inserted into his left arm.

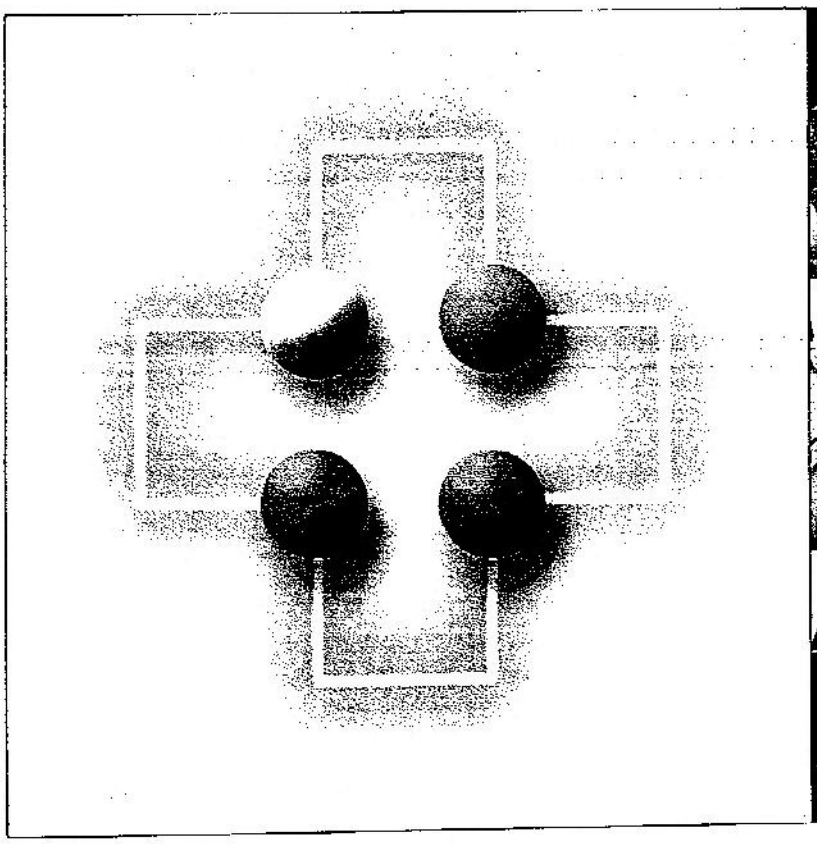
What is the provisional diagnosis?
The provisional diagnosis is asthma

Young aged female conscious patient sitting comfortable in her bed, thin, neither dyspneic and nor tachypneic. She had pale palmer creases, koilonychia involving all of her digits, with no digital clubbing. Both radial and brachial arteries were patent; the radial pulse was 100 beats per minutes, of large volume, and regular rhythm. She had facial pallor, blue sclera, and no jaundice. She had angular stomatitis with depapillated tongue; bad oral hygiene with bad breath smell. Her JVP was not distended, the trachea was centrally located, and there was no goiter or lymphadenopathy. There was no leg edema, and both dorsalis pedis and posterior tibial arteries were patent. She was supplied with pint of blood infusing through giving set into black colored cannula.

What is the provisional diagnosis?
The provisional diagnosis is anemia (IDA)

3

VITAL SIGNS



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VITAL SIGNS MEASUREMENT

Vital signs include the assessment of blood pressure, body temperature, respiratory rate, radial pulse, and pulse oximetry.

BLOOD PRESSURE ASSESSMENT

Accurately measured blood pressure is paramount in the assessment and treatment of hypertension. Aneroid manometers are less accurate than mercury manometers but are sufficient if frequently standardized against a mercury column. Blood pressure should be measured two or three times after the patient has been seated comfortably for at least 5 minutes, the back supported, and the arm bare and at the heart level. The readings should then be averaged. Tobacco and caffeine should be avoided for at least 30 minutes. In adults blood pressure more than 140/90 mmHg is considered hypertension. Hypertension can be diagnosed on the basis of one reading if it is associated with target organ damage or is higher than 220/120 mmHg. Falsely high blood pressure readings occur when the cuff is too narrow or when the bladder is too short to encircle the arm. Three abnormal readings should be present, preferably over a period of several weeks, before treatment is begun. However hypertension should be treated immediately when any one of the following is present:

1 Hypertension with end organ damage
   Hypertensive retinopathy (grade III-IV)
   Hypertensive left ventricular failure
   Hypertensive encephalopathy
   Intracranial hemorrhage
   Rapidly progressive renal failure
2 Severe hypertension (>220/>120)
3 Dissecting aortic aneurysm
4 Sever preeclamsia

The British Hypertension Society has defined ranges of blood pressure which fall within the normal range and those that indicates hypertension (BOX 3 – 1).

BOX 3 – 1. Definition of hypertension		
Category	Systolic blood pressure	Diastolic blood pressure
BLOOD PRESSURE		
Optimal	< 120 mmHg	< 80 mmHg
Normal	< 130 mmHg	< 85 mmHg
High normal	130-139 mmHg	85-89 mmHg
HYPERTENSION		
Grade 1 (mild)	140-159 mmHg	90-99 mmHg
Grade 2 (moderate)	160-179 mmHg	100-109 mmHg
Grade 3 (severe)	≥180 mmHg	≥110 mmHg
ISOLATED SYSTOLIC HYPERTENSION		
Grade 1	140-159 mmHg	< 90 mmHg
Grade 2	≥ 160 mmHg	< 90 mmHg

In most cases the causes of hypertension is not known and the term essential hypertension is applied but in about 5% of cases, secondary hypertension is said to be present (BOX 3 – 2).

BOX 3 – 2. Causes of hypertension		
Essential or primary hypertension		
Secondary hypertension with an underlying cause (MNEMONIC APCDE-OR)	• Alcohol • Pregnancy induced hypertension • Coarctation of aorta • Drugs e.g. NSAIDs, steroid, and contraceptive pills	• Endocrine disease e.g. Cushing's syndrome, acromegaly and pheochromocytoma • Obesity • Renal diseases e.g. renal artery stenosis, glomerulonephritis and polycystic kidney

Secondary hypertension should be clinically considered when:

1 The age of onset is less than 35 years or more than 55 years
2 Hypertension is difficult to control after therapy has been instituted
3 Hypertension is accelerated or malignant
4 Signs of secondary hypertension are present

CLINICAL CLUE

The classic triad of sudden severe headaches, diaphoresis, and palpitations carries a high degree of specificity (94%) and sensitivity (91%) for pheochromocytoma in hypertensive patients. The absence of all three symptoms reliably excludes the condition.

Measurement of the blood pressure should be made to the nearest two mmHg and the following steps are suggested:

1 STEP I Use a machine that has been validated, well maintained and properly calibrated
2 STEP II Remove tight clothing from the arm. You can measure over thin clothing, as it makes no difference to the result
3 STEP III Support the arm at the level of the heart.

CLINICAL CLUE

If the arm is 7 cm higher than the heart level, both systole and diastole pressures will be 5 mmHg lower. If the arm is 7 cm lower than the heart, they will be about 6 mm higher

4 STEP IV Use a cuff with appropriate size (the bladder must encompasses > two third-third of the arm). It is usually of a width of 12 cm with the lower border 2.5 cm from cubital fossa
5 STEP V Palpate the brachial pulse first and then the radial pulse and inflate cuff until radial pulse is impalpable.
6 STEP VI Inflate the cuff another 30 mmHg and listen through the diaphragm of the stethoscope placed over the brachial artery.

CLINICAL CLUE

The stethoscope should be applied lightly without exerting excess pressure. Excess pressure can artificially lower the diastolic reading by 10 mmHg. The systolic pressure is not usually affected

7 STEP VII Deflate slowly (2 mm per second) until the radial pulse becomes palpable and a regular tapping (phase 1 Korotkoff sounds) sound is heard which is normally in adult 100 –140 mmHg. This corresponds to the systolic blood pressure.

CLINICAL CLUE

Up to 20% of elderly hypertensive patients have phase 1 Korotkoff sounds which begin at systolic pressure but then disappear for varying lengths of time, reappearing before diastolic pressure. This is termed auscultatory gap. If the first appearance of the sound is missed, the systolic pressure will be recorded at a falsely low level. Avoid this by palpating the systolic pressure via radial pulse first

8 STEP VIII As the pressure of the cuff continues to fall, korotkoff sound will be muffled (phase 4) then disappeared (phase 5). The disappearance corresponds to the diastolic pressure which is normally in adults 60 – 90 mmHg.

CLINICAL
CLUE

Occasionally, muffled sounds persist and do not disappear, in which case the point of muffling is the diastolic pressure. In adults blood pressure of more than 140/90 mmHg is considered as hypertension. Prehypertension is a new designation for mildly elevated blood pressures between 120/80 and 139/89 mm Hg, a level at which progression to hypertension is twice as likely as with a blood pressure below 120/80 mm Hg

9 STEP IX Blood pressure should be measured in both arms. A difference of <10 mmHg is considered normal but the highest reading is taken for assessment. A difference >10 mmHg is considered abnormal

10 STEP X In children and young adults (less than 35 years) suffering of hypertension, the blood pressure should be measured in legs and arms. In coarctation of the aorta, normal or low blood pressure in the legs, but hypertension in the arm.

CLINICAL
CLUE

Blood pressure discrepancy between the arms (> 10 mmHg) or between the arms and legs should raise the possibility of coarctation of aorta, arterial occlusion or stenosis of any cause, persistent patent ductus arteriosus, dissecting aortic aneurysm, thoracic outlet syndrome, and supravalvular aortic stenosis

11 STEP XI Blood pressure should be measured in both arms and after 3 minutes of standing to exclude a significant postural fall in blood pressure, particularly in older persons and in those with diabetes or other conditions (e.g., Parkinson's disease) that predispose to autonomic insufficiency.

CLINICAL
CLUE

Postural hypotension is defined as reduced in systolic blood pressure more than 20 mmHg and/or reduced diastolic blood pressure more than 15 mmHg after 3 minutes of standing from supine position

Blood pressure measurement therefore taken as shown in FIGURE 3 – 1.

FIGURE 3 – 1. The techniques for blood pressure measurement includes palpation of the brachial artery (A), palpation of the radial artery before inflation of the blood pressure cuff (B), and then inflation of the cuff till the radial pulse becomes impalpable. After that, inflate the cuff for further 30 mmHg and deflate slowly till the radial pulse become palpable again and you hear tapping sound through the brachial artery (C). This is the systolic blood pressure. Disappearance of the sound corresponds to the diastolic blood pressure.

The pulse pressure is the difference between the systolic and diastolic pressures. A pulse pressure of ≥80 mmHg suggests aortic regurgitation, while a low pulse pressure may occur in aortic stenosis. Emergent hypertension is an acute, often severe, elevations in blood pressure, accompanied by acute (or rapidly progressive) target organ dysfunction, such as myocardial or cerebral ischemia or infarction, pulmonary edema, or renal failure. Urgent hypertension is a severe elevation in blood pressure without severe symptoms and without evidence of acute or progressive target organ dysfunction. Emergent hypertension requires immediate intensive care unit (ICU) admission for intravenous therapy and continuous blood pressure monitoring; urgent hypertension often can be managed with oral medications and appropriate outpatient follow-up in 24 to 72 hours. Resistant hypertension is defined as blood pressure that remains above goal despite treatment with the optimal dosages of three antihypertensive agents of different classes, including a diuretic.

BODY TEMERATURE ASSESSMENT

The warmth of the skin to the touch may provides a good indication of fever, but the skin of a patient with a normal temperature may be felt cold, and an apparently normal skin temperature does not exclude hypothermia. The normal body temperature measurement is 36.8 ± 0.4°C (BOX 3 – 3).

BOX 3 – 3. Ranges of body temperature

	Centigrade	Fahrenheit
Normal	36.4 – 37.2	98 – 99
Subnormal	< 36.4	< 98
Febrile	a.m. > 37.2	> 99
	p.m. > 37.7	> 100
Hyperpyrexia	> 41.5	> 107
Hypothermia	< 35	< 95

Six sites are used to measure body temperature. These are:

1	Mouth (oral)	See BOX 3 - 4
2	Rectum	Thermometer is inserted into the rectum
3	External auditory meatus	
4	Forehead	
5	Axilla	Thermometer is left for two minutes
6	Groin	In the fold of the groin and thigh flexed on to the abdomen

The temperature in axilla or groin is about 0.5 °C lower than oral temperature and the rectal temperature is about 0.5°C higher than oral temperature. In infants and young children < 5 years, use the axilla or groin as a site for temperature measurement, but in older children > 5 years and conscious adults, use the oral method. Moreover, in collapsed or comatose patients, use the rectal method. Temperature in infants is about normally higher than that of older children 1 °C.

BOX 3 – 4. Recommendations in measuring oral temperature

The thermometer must be accurate
Wash the thermometer with antiseptic solution and shake it before and after use
Should be left for minute in position
The patient must breathe through the nose and keep the lips firmly closed during the observation

Fever is generally accompanied by tachycardia, with the general rule of thumb that the heart rate will increase by 10 beats per minute for every 1 C° increase in temperature. The absence of tachycardia with fever is known as pulse-temperature dissociation or relative bradycardia and has been reported in typhoid fever, brucellosis, leptospirosis, some drug-induced fevers, and factitious fever.

RESPIRATORY RATE ASSESSMENT

The normal rate of respiration in a relaxed adult is about 14 – 18 breaths (cycles) per minute. It is advisable to count the respiratory rate while your hand at the radial pulse so as to withdraw the attention of the patient. Respiratory rate more than 20 breaths (cycles) per minute is called tachypnea which may be caused by increased ventilatory drive in fever, acute asthma and exacerbation of COPD, or reduced ventilatory capacity in pneumonia, pulmonary edema and interstitial lung disease. Respiratory rate less than 10 breaths (cycles) per minute is called bradypnea which occurs in opioid toxicity, hypercapnia, hypothyroidism, raised intracranial pressure and hypothalamic lesions. Complete cessation of respiration is called apnea, which occurs in voluntary holding of breath, Cheyne-Stokes breathing, and during sleep (obstructive sleep apnea and central sleep apnea).

RADIAL PULSE ASSESSMENT

Radial pulse is obtained from radial artery which is felt with the tips of the index, middle and ring finger compressing the vessel against the lower end of the radius (FIGURE 3 – 2). The patient's forearm should be slightly pronated and the wrist slightly flexed. The radial pulse can be absent in certain pathological conditions (BOX 3 – 5). When taking a pulse, characterize the information according to:

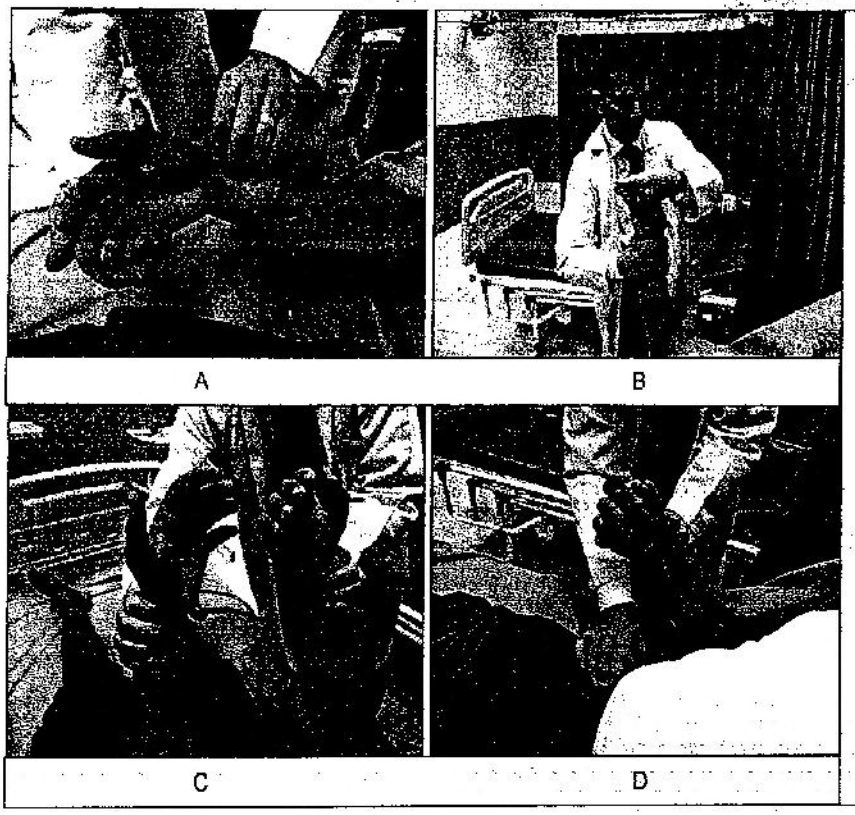


FIGURE 3 – 2. The techniques for radial pulse examination should include palpation of the radial pulse (A), detection of the collapsing pulse (B), looking for radioradial delay (C), and radiofemoral delay (D).

BOX 3 – 5. Some pathological causes of absent radial pulse	
Congenital anomalies	Iatrogenic: post-catheterization and following a radial artery line for monitoring of blood gases or arterial pressure
Dissection of aorta with subclavian involvement	Trauma
Embolism into the radial artery	Takayasu's arteritis

THE PULSE RATE

The rate of the radial pulse is noted in beats/minute. Count the pulse rate over 15 seconds and multiply by 4 to obtain the beats per minute (bpm). It is normally 60 – 100 beats per minute. Pulse rate (BOX 3 – 6) more than 100 bpm is called tachycardia and that less than 60 bpm is called bradycardia.

BOX 3 – 6. Some pathological causes of bradycardia and tachycardia	
Sinus bradycardia	Sinus tachycardia
Myocardial infarction (especially inferior wall)	Anxiety
Heart block e.g. complete heart block	Fever
Hypothermia	Anemia
Hypothyroidism	Heart failure
Cholestatic jaundice	Thyrotoxicosis
Raised intracranial pressure	Pheochromocytoma
Drugs, e.g. β-blocker, digoxin, verapamil	Drugs, e.g. β-adrenoceptor agonists, theophylline

If you are in any doubt about whose pulse you are feeling, palpate your own pulse at the same time. If what you are feeling is not synchronous with yours it is the patient's pulse.

THE RHYTHM

The rhythm can be described as either regular or irregular rhythm (BOX 3 – 7). The latter is further divided into:

1 Irregular irregularity (completely irregular pattern) as may occur in atrial fibrillation
2 Regular irregularity (recurring irregular pattern) as may occur in bigeminy (alternating ectopic and normal beat) and trigeminy (alternating ectopic and two normal beats)

BOX 3 – 7. Some pathological causes of irregular radial pulse

Sinus arrhythmia	Atrial fibrillation
Atrial extrasystoles	Atrial flutter with variable response
Ventricular extrasystoles	Second degree heart block with variable response

THE VOLUME

Volume of the radial pulse reflects the left ventricular stroke volume. The volume is described as good volume, large volume (BOX 3 – 8), or small volume. A small pulse volume may be due to reduced stroke volume and occurs in left ventricular failure, hypovolemia or peripheral vascular disease.

BOX 3 – 8. Some causes of increased pulse volume

Physiological	Pathological	
Exercise	Patent ductus arteriosus	Aortic regurgitation
Pregnancy	Fever	Paget's disease of bone
Increased environmental temperature	Thyrotoxicosis	Peripheral AV shunt
Advanced age	Anemia	

THE CHARACTER

The character is poorly assessed by palpating the radial pulse; the carotid or brachial arteries should be used for this purpose as these more accurately reflect the form of the aortic pressure wave. However, the collapsing (bounding) pulse, pulsus bigeminus, and pulsus alternans may be readily apparent in the radial pulse. Collapsing pulse or water hammer sign is a pulse with rapid upstroke and descent (pulse which feels as though it suddenly hits your fingers and falls away just as quickly). The upstroke owing to increased stroke volume while the descent is due to flowing of blood into the left ventricle during diastole and due to low systemic vascular resistance. To feel collapsing pulse (FIGURE 3 – 2), raise the arm using left hand while you feel the pulse using your right hand. This pulse can occur in aortic regurgitation and may also occur in hyperdynamic circulation. Ventricular ectopic beats are usually followed by a compensatory diastolic pause which allows increased ventricular filling, and produces a large stroke volume in the sinus beat following the ectopic beat. Sometimes ectopic beats occur regularly, e.g. after every normal beat. This is called pulsus bigeminus. Pulsus alternans means alternating strong and weak pulsation that occurs due to advanced left ventricular failure. A blood-pressure cuff gives you a more sensitive method. After raising the cuff pressure, lower it slowly to the systolic level and then below it a few millimeters of mercury. While you do this, the patient should breathe quietly or stop breathing in the respiratory midposition. If dyspnea prevents this, help patient to sit up and dangle both legs over side of bed. Loud and soft Korotkoff sounds or a sudden doubling of apparent heart rate as the cuff pressure declines indicates a pulsus alternans.

WHAT IS MEANT BY PULSUS PARADOXUS?

Pulsus paradoxus is an exaggeration of the normal variability of pulse volume with the respiratory cycle. Pulse volume normally increases in expiration and decreases during inspiration due to intrathoracic pressure changes affecting venous return to the heart. The technique for paradoxical pulse is as follows: Have the patient breathe as normally as possible. Inflate the blood pressure cuff until no sounds are heard. Gradually deflate the cuff until sounds are heard in expiration only. Note this pressure. Continue to deflate the cuff slowly until sounds are heard during inspiration. Note this pressure. If the difference in these two pressures exceeds 10 mm Hg, a marked (abnormal) pulsus paradoxus is present; cardiac tamponade may be the cause. It is not a specific phenomenon for tamponade because it is also seen in large pericardial effusions, in constrictive pericarditis, and in conditions associated with increased ventilatory effort, such as asthma and emphysema.

RADIOFEMORAL DELAY

Simultaneously feel the radial pulse and the femoral pulse (FIGURE 3 – 2). If you have found that the radial pulse comes before the femoral pulse (femoral delay), diagnosis of coarctation of aorta will probably be made. It is a characteristic of the coarctation of the aorta; in which, there is narrowing of the aorta just beyond the origin of the left subclavian artery.

RADIORADIAL DELAY

It can also be useful to palpate both radial pulses together to detect radial-radial inequality in timing or volume. This condition occurs due to arterial occlusion by an atherosclerotic plaque, subclavian artery stenosis, dissecting aortic aneurysm, or due to thoracic outlet syndrome.

PULSE DEFICIT

When the rhythm of the pulse is irregularly irregular (which indicates atrial fibrillation) you have to count the heart rate by auscultation. The difference between heart rate and pulse rate is called pulse deficit.

PULSE DEFICIT = HEART RATE – PULSE RATE

CONDITION OF THE VESSEL WALL

Degenerative changes in arteries that make them less elastic are referred to collectively as arteriosclerosis (hardening of the arteries). Obesity, high dietary cholesterol and smoking are some of the factors correlated with premature development of arteriosclerosis. The status can be therefore described as normal elastic state or as a rigid hard state especially in the elderly.

PULSE OXIMETRY

Pulse oximetry is a simple method (FIGURE 3 – 3) of obtaining the oxygen saturation (Sa_{O2}). The pulse oximetry consists of a probe attached to the patient's finger which is linked with a computerized unit. The oxygen saturation should always be above 95%. An oxygen saturation of < 92% indicates hypoxia and the need for oxygen therapy. An oximeter detects hypoxia before the patient becomes clinically cyanosed. In a number of situations the pulse oximeter may not be accurate. Whenever the flow is sluggish e.g. hypovolemic shock, cardiogenic shock, or in case of vasoconstriction, the oximeter may not function. Hypothermia causes reduced tissue perfusion and failure to register a signal. Dyes and nail varnish may give artificially low values. Dark skin pigmentation and raised levels of bilirubin can result in falsely high value. Cardiac valvular diseases e.g. tricuspid regurgitation can rarely cause venous pulsation and therefore venous oxygen saturation is recorded by the oximeter. Cardiac arrhythmia may interfere with the oximeter picking up the pulsatile signal properly and with the calculation of the pulse rate. The presence of methemoglobin will result in falsely low oxygen saturation but normal arterial oxygen tension. Carboxyhemoglobin causes saturation values towards 100% but normal arterial oxygen tension (BOX 3 – 9). A pulse oximetry therefore is extremely misleading in case of carbon monoxide poisoning. A common finding in patients with markedly elevated immature white blood cells counts e.g. leukemia is low arterial oxygen tension on arterial blood gas with a normal pulse oximetry.

FIGURE 3 – 3. Detection of oxygen saturation by pulse oximetry.

BOX 3 – 9. Some causes of abnormal oxygen saturation or tension

	Clinical condition	Oxygen saturation by pulse oximetry	Arterial oxygen tension by blood gas analysis
1	Hypoxia	Low (below 92%)	Low (below 12 Kpa)
2	Methemoglobin	Low (about 85%)	Normal
3	Carboxyhemoglobin	High	Normal
4	Acute leukemia	Normal function	Low due to consumption

EXAMPLES

The blood pressure was 120 over 80 millimeter mercury in supine position of both arms; the pulse was 70 beats per minutes, regular, good volume, with no collapsing pulse, no radioradial or radiofemoral delay, and soft vessel walls; temperature is 37.5 corrected axillary method (i.e. measured at the axilla and 0.5 °C is added); respiratory rate is 15 cycles per minute; the oxygen saturation is 96%.

What is the interpretation?
The interpretation is normal vital signs

The blood pressure is 100 over 60 millimeter mercury in supine position and 70 over 40 in standing; the pulse is 80 beats per minutes that increased to 100 beats per minute on standing, regular, very small volume, with no collapsing pulse, no radioradial or radiofemoral delay, and soft vessel walls; temperature is 37.5 °C corrected axillary method; respiratory rate is 22 cycles per minute; the oxygen saturation can not be detected.

What is the interpretation?
The interpretation is shocked state

The blood pressure is 100 over 60 millimeter mercury in supine position and 70 over 40 in standing; the pulse is 80 beats per minutes on supine and standing, regular, good volume, with no collapsing pulse, no radioradial or radiofemoral delay, and hard vessel walls; temperature is 37.5 °C orally; respiratory rate is 15 cycles per minute; the oxygen saturation is 96%.

What is the interpretation?
The interpretation is autonomic dysfunction e.g. diabetes. The heart rate fails to increase in response to dropping of blood pressure indicating autonomic disturbance rather than hypovolemia.

The blood pressure is 160 over 110 millimeter mercury in supine position and standing position; the pulse is 75 beats per minutes, irregularly irregular, variable volume, with no collapsing pulse, no radioradial or radiofemoral delay, and hard vessel walls; temperature is 37.5 °C orally; respiratory rate is 15 cycles per minute; the oxygen saturation is 95%.

What is the interpretation?
The interpretation is atrial fibrillation due to hypertension.

The blood pressure is 140 over 90 millimeter mercury in supine position of the right arm but very faint on the left; the pulse is 95 beats per minutes on the right, irregularly irregular, variable volume, with no collapsing pulse, there is radioradial inequality, no radiofemoral delay, and hard vessel walls; temperature is 38.5 °C orally; respiratory rate is 22 cycles per minute; the oxygen saturation is 95% on the right but can not be detected on the left.

What is the interpretation?
The interpretation is limb ischemia (left) due to atrial fibrillation. High temperature is due to tissue damage.

The blood pressure is 100 over 90 millimeter mercury in supine and standing position; the pulse is 65 beats per minutes, regular, good volume, with no collapsing pulse, no radioradial or radiofemoral delay, and hard vessel walls; temperature is 39.5 °C orally; respiratory rate is 22 cycles per minute; the oxygen saturation is 98%.

What is the interpretation?
The interpretation is febrile disorder with relative bradycardia.

SYSTEMIC EXAMINATION

This chapter is designed to be used by fourth medical students and elder students as far as postgraduate doctors who may have forgotten what they learned as students. It is universally recommended that the approach is undertaken under four fundamental elements. These are:

1. Inspection — It is what you see
2. Palpation — It is what you feel
3. Percussion — It is what you hear, or perceive, by percussion
4. Auscultation — It is what you hear by a stethoscope

This rule is applied for all systemic examination apart from nervous and locomotor systems. This chapter is divided into five sections. The first section deals with the cardiovascular system; the second section deals with the respiratory system; the third section deals with the abdomen examination that involves both the gastrointestinal system and the genitourinary system; the fourth section deals with the nervous system examination; and the fifth section deals with the locomotor system. Third medical students need not to read theses sections. It is generally accepted that their clinical duty is to perform clinical history with/without general examination. Fourth medical students should cover all the initial four sections. The plan in Iraq is to perform systemic examination involving the cardiovascular system, the respiratory system, examination of the abdomen, and examination of the nervous system. This plan is taken together with general examination. Fifth medical students should in addition learn how to examine the locomoter system while the sixth medical students should completely cover this chapter. Postgraduate doctors may approach piecemeal as seems appropriate.

Every section in this chapter is discussed under two parts; the anatomical landmarks and the physical examination. The systemic approach of the cardiovascular system implies examination of the cardio (precordium) and the vascular (vessels), but this rule is not always applicable in clinical training. It is generally recommended to firstly make approach to the general examination peculiar to the cardiovascular system and then to examine the precordium. Some examiners however prefer the reverse style.

THE ORDERS OF EXAMINATION

In short case examination, the order is to examine either the whole system or single element of that system. Due to the shortage of time, the examination is sometimes ordered to single part:

1. Examine the tone of the lower limbs
2. Auscultate the heart
3. Examine the cervical lymph nodes
4. Examine for visceromegaly
5. Palpate the abdomen
6. Examine the facial nerve
7. Auscultate the back

Every student should ask his/her senior trainer about the order of examination during clinical training since every examiner has his/her own approach. This variability in the approach reflects really the variability of the guidelines that senior trainer follows.

THE PRESENTATION

The presentation of the clinical findings in any clinical setting is performed under two ways:

1. Running commentary presentation
2. Deferred commentary presentation

During clinical training the best way for the students is the first presentation. In which you should orally present the techniques and the findings whilst the process of examination continues. In the second one, you should firstly examine the patient and then present your findings. The second presentation is the commonest examination technique. The time given for the candidate during the examination is variable and ranges from three to five minutes. The candidate should therefore learn him/her self for that.

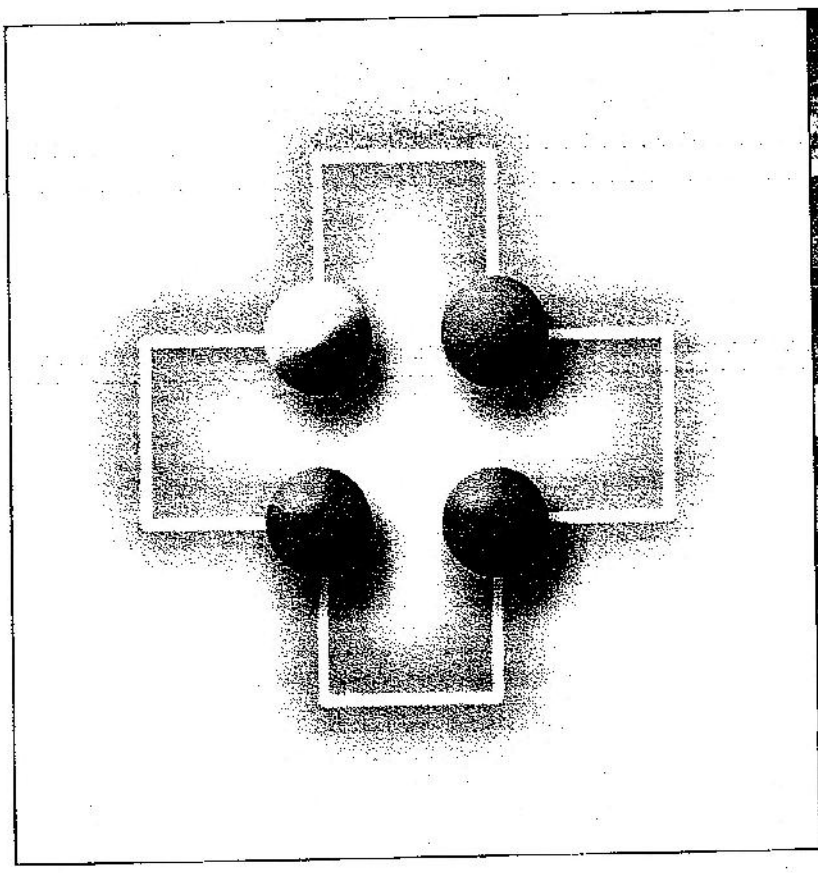
COMMON CLINICAL MISTAKES

Some of the common clinical mistakes during examination are:

1. The history and/or the examination is performed on the left side of the bed
2. The student records a diagnosis or a medically correct terms in the history of present illness
3. The student is not fully equipped at the time of examination
4. The student omits an element of the examination

4

CARDIOVASCULAR SYSTEM



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ANATOMY OF THE CARDIOVASCULAR SYSTEM

The heart has the shape of a blunt cone and is approximately the size of a closed fist. It is in the thoracic cavity between the lungs in a space within the mediastinum called the pericardial cavity. The blunt, rounded point of the cone is the apex, and the larger, flat portion at the opposite end of the cone is the base. The heart lies obliquely in the mediastinum with its base directed anteriorly and slightly inferiorly. The apex is also directed to the left so that approximately two thirds of the heart's mass lies to the left of the midline of the sternum. The heart consists of four chambers; two atria and two ventricles. The heart of healthy 70 kg person pumps approximately 7200 liters of blood each day at a rate of 4 – 8 liters per minute.

Seven large veins carry blood to the heart; four pulmonary veins carry blood from the lungs to the left atrium; the superior and inferior venae cavae carry blood from the body to the right atrium; and the coronary sinus carries blood from the heart's wall to the right atrium. Two arteries, the aorta and the pulmonary trunk, exit the heart. The aorta carries blood from the left ventricle to the body; the pulmonary trunk carries blood from the right ventricle to the lungs.

RIGHT AND LEFT ATRIA

The right atrium has three major openings; the superior vena cava, the inferior vena cava, and the coronary sinus. The left atrium has four relatively uniform openings that receive the four pulmonary veins. The two atria are separated from each other by the interatrial septum. A slight oval depression, the fossa ovalis on the right side of the septum makes the former location of the foramen ovale, an opening between the right and left atria in the embryo and the fetus.

RIGHT AND LEFT VENTRICLES

The atria open into the ventricles through atrioventricular canals. The two ventricles are separated from each other by the interventricular septum which is thick (the muscular portion) toward the apex and very thin (the membranous portion) toward the atria. The right ventricle opens into the pulmonary trunk, and the left ventricle opens into the aorta.

ATRIOVENTRICULAR VALVES

An atrioventricular valve is on each atrioventricular canal and is composed of cusps, or flaps. These valves allow blood to flow from the atria into the ventricles but prevent blood from flowing back into the atria. The valve between the right atrium and the right ventricle has three cusps and is called the tricuspid valve. The valve between the left atrium and left ventricle has two cusps and is called the bicuspid, or mitral valve.

Each ventricle contains cone-shaped muscular pillars called papillary muscle. These muscles are attached by thin, strong connective tissue strings called chordae tendineae to the cusps of the atrioventricular valves.

The papillary muscles contract when the ventricles contract and prevent the valves from opening into the atria by pulling on the chordae tendineae attached to the valve cusps. Blood flowing from the atrium into the ventricle pushes the valve open into the ventricle, but when the ventricle contracts blood pushes the valve back toward the atrium. The atrioventricular opening closes as the valve cusps meet.

SEMILUNAR VALVES

The aorta and pulmonary trunk possess aortic and pulmonary semilunar valves. Each valve consists of three pocket-like semilunar cusps.

AORTA

All arteries of the systemic circulation are derived either directly or indirectly from the aorta, which usually is divided into three general portions; the ascending aorta, the aortic arch, and the descending aorta. The descending aorta is divided further into a thoracic aorta and an abdominal aorta.

At its origin from the left ventricle, the aorta passes superiorly from the heart. This portion is called the ascending aorta. It has only two arteries branching from it, the right and left coronary arteries, which supply blood to the cardiac muscle. The aorta then arches posteriorly and to the left as the aortic arch. The first vessel to branch from the aortic arch is the brachiocephalic artery that branches at the level of the clavicle to form the right common carotid artery which transports blood to the right side of the head and the neck and the right subclavian artery which transports blood to the right upper limb. The second branch of the aortic arch is the left common carotid artery which transports blood to the left side of the neck. The third branch of the aortic arch is the left subclavian artery which transports blood to the left upper limb. The common carotid arteries branch at the inferior angle of the mandible into internal carotid artery and external carotid artery.

The next portion of the aorta is the descending aorta. It is the longest portion of the aorta and extends through the thorax on the left side of the mediastinum and through the abdomen to the superior margin of the pelvis. The thoracic aorta is that portion of the descending aorta located in the thorax. It has several branches that supply various structures between the aortic arch and the diaphragm. The abdominal aorta is that portion of the descending aorta between the diaphragm and the point at which the aorta ends by dividing into the two common iliac arteries.

The abdominal aorta has several branches. Its terminal branches, the common iliac arteries, supply blood to the pelvis and the lower limbs.

EXAMINATION OF THE CARDIOVASCULAR SYSTEM

Generally speaking the sequence of the cardiovascular system examination is undertaken under two steps:

1 General examination peculiar to the cardiovascular system
2 Examination of the precordium

GENERAL EAXMINATION PECULIAR TO THE CARDIOVASCULAR SYSTEM

As we have learned in the general examination the steps include the general look and the regional examination. The general look should include age group, sex, awareness, decubitus, state of comfortability, body built, and dyspnea and tachypnea. The regional examination on the other hand should include examination of the hands, face, neck, lower limbs, and peripheral pulses. These should be interpreted with particular association to the cardiovascular system.

THE HANDS (FIGURE 4 – 1)
Look for clubbing, splinter haemorrhages, Osler's nodes, Janeway lesions, xanthomas, koilonychia, etc. Quincke's sign is a capillary pulsation in the nail bed due to aortic valve regurgitation. It is also called nail bed pulsation.

THE FACE (FIGURE 4 – 1)
Anaemia may reflect haemodynamic instability manifested as pale conjunctiva. Like wise hyperlipidaemia is manifested as xanthelasma.

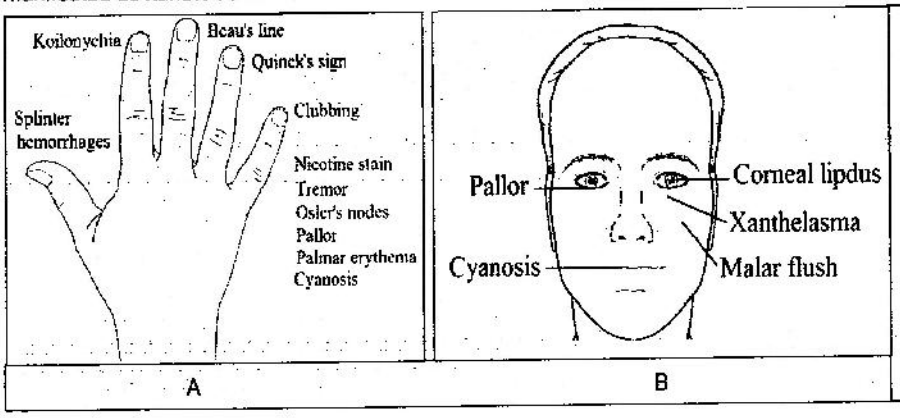


FIGURE 4 – 1. Some clinical hand (A) and facial (B) signs in cardiovascular diseases.

THE NECK
Examine the vessels, thyroid, trachea, and lymphadenopathy as we discussed that earlier.

THE LEGS (FIGURE 4 – 2)
Examination of the legs may reveal evidence of the peripheral vascular disease including both arterial disorder in the form of limb ischemia and venous disorder in the form of DVT, varicose veins as well as for both arterial and venous ulceration. Examination should also include edema.

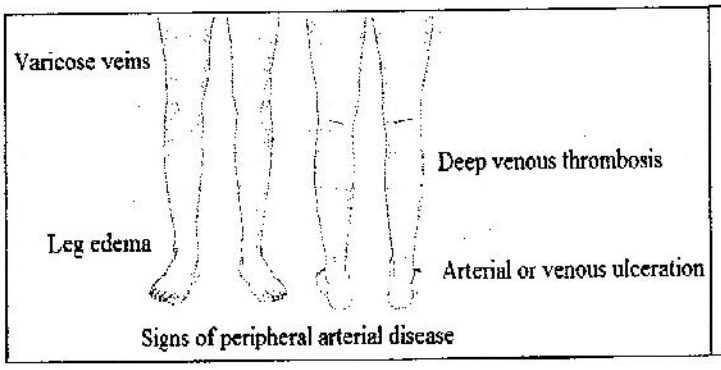


FIGURE 4 – 2. Some clinical leg signs in cardiovascular diseases.

EXAMINATION OF THE PERIPHERAL VESSELS
Radial and brachial arteries had been discussed in details earlier. Other arteries will be discussed here. In general the peripheral arterial pulses are sometimes recorded as:

+ Normal pulse
± Reduced pulse
– Absent pulse
++ Aneurismal pulse

CAROTID ARTERY

The patient's right carotid artery is examined by gentle compression against the transverse processes of the cervical vertebrae with the candidate's left hand (FIGURE 4 – 3). Try not to compare right with left carotid artery at the same time as this may induce syncope. Carotid artery is better than radial artery in description of the volume and character of blood flow. Anacrotic pulse is slow rising pulse with a palpable shudder and occurs in aortic stenosis. It is also called parvus and tardus pulse. Bisferiens pulse (pulsus bisferiens) is a wave form with two peaks. It is the combination of anacrotic pulse and collapsing pulse and is a characteristic of aortic regurgitation (with or without accompanying aortic stenosis) and of hypertrophic cardiomyopathy. Jerky pulse is a pulse that may feel normal at first but peter out quickly. It is a characteristic of hypertrophic obstructive cardiomyopathy. Dicrotic pulse results from an accentuated dicrotic wave and tends to occur in patients with septic shock, cardiogenic shock, hypovolemic shock, obstructive shock due to cardiac tamponade, and in patients with aortic valve replacement.

FIGURE 4 – 3. Examination of the carotid artery.

FEMORAL ARTERY

It is felt in the midway between symphysis pubis (midline) and anterior superior iliac spine (ASIS) in the right groin using the pulps of the right index, middle and ring fingers. It is also used to describe the volume of the blood flow. Compare after that with the contralateral left femoral artery at the same time. The mnemonic NAVEL may help you remember the lateral to medial sequence of Nerve Artery Vein Empty space Lymph node.

POPLITEAL ARTERY

It is difficult to feel because it does not cross a prominent bone and is not superficial (FIGURE 4 – 4). Four tests are used to describe volume of blood flow in the popliteal artery:

1. TEST I Flex knee to 135°, place the thumbs on the tibial tuberosity and the fingers over the lower part of the popliteal fossa (FIGURE 4 – 4 A)
2. TEST II Flex knee to 90°, sit beside the patient and compress the fingers against medial condyle of the femur
3. TEST III Make the patient in prone position and feel the artery with the fingers' tips of both hands
4. TEST VI The crossed leg test or the popliteal pulsating test. When a person sits with the legs crossed, an oscillatory movement of the foot will occur if the popliteal artery is intact (FIGURE 4 – 4 D and E)

The posterior tibial artery, anterior tibial artery, and dorsalis pedis artery are discussed earlier.

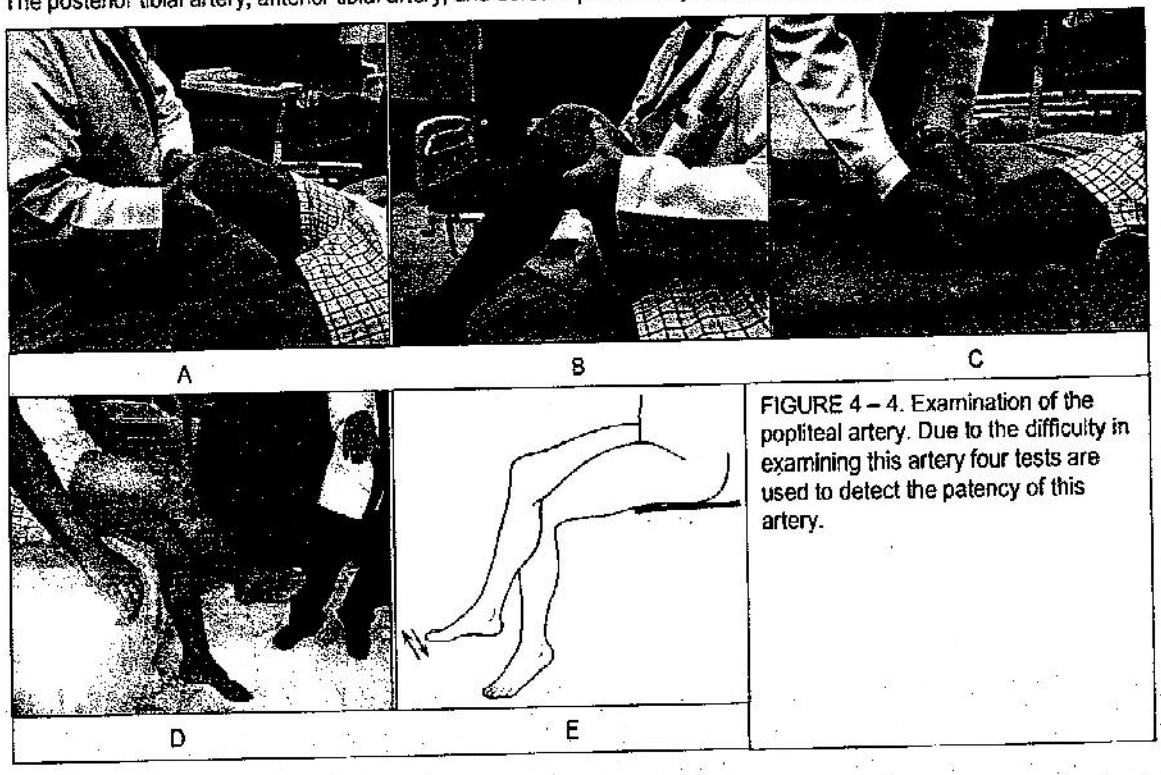


FIGURE 4 – 4. Examination of the popliteal artery. Due to the difficulty in examining this artery four tests are used to detect the patency of this artery.

SPECIAL TECHNIQUE/ULNAR ARTERY

If you suspect arterial insufficiency in the arm or hand, try to feel the ulnar pulse (FIGURE 4 – 5). Feel for it deeply on the flexor surface of the wrist medially. The pulse of a normal ulnar artery, however, may not be palpable. The Allen test (FIGURE 4 – 6) gives further information. This test is also useful to assure the patency of the ulnar artery before puncturing the radial artery for blood samples. The patient should rest with hands in lap, palms up. Ask the patient to make a tight fist with one hand; then compress both radial and ulnar arteries firmly between your thumbs and fingers. Next, ask the patient to open the hand into a relaxed, slightly flexed position. The palm is pale. Release your pressure over the ulnar artery. If the ulnar artery is patent, the palm flushes within about 3 to 5 seconds. Patency of the radial artery may be tested by releasing the radial artery while still compressing the ulnar.

FIGURE 4 – 5. Ulnar artery detection

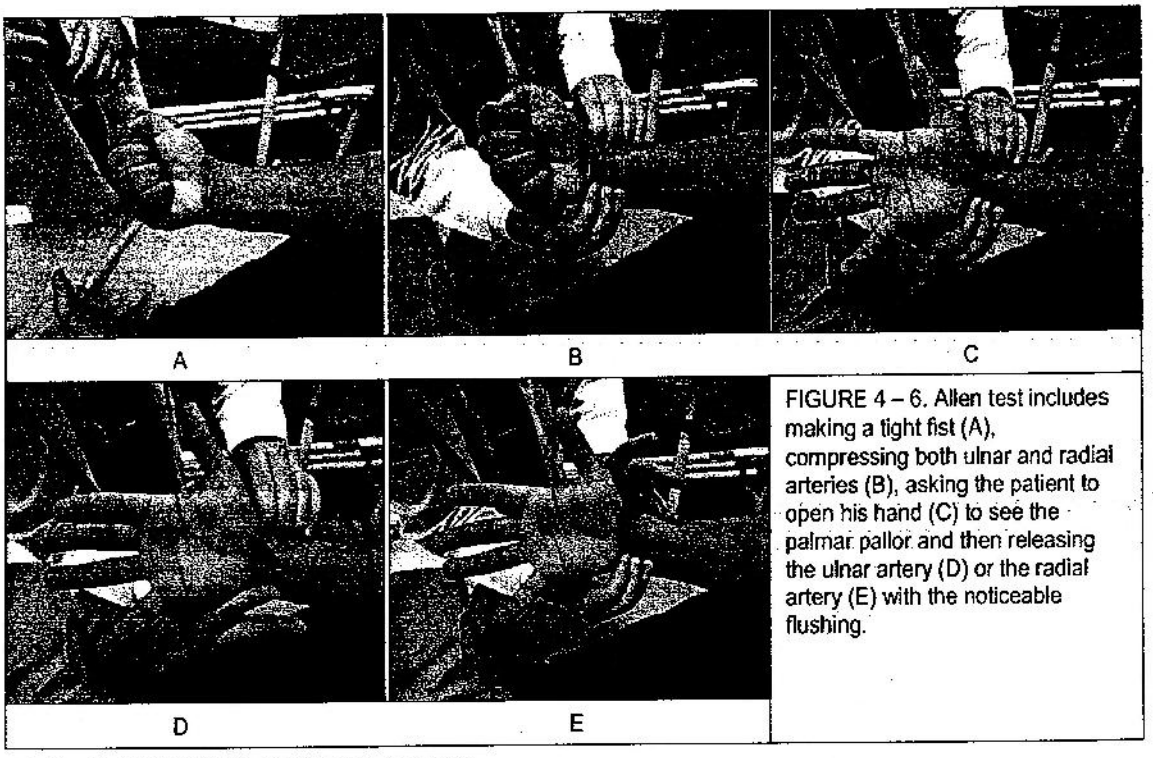


FIGURE 4 – 6. Allen test includes making a tight fist (A), compressing both ulnar and radial arteries (B), asking the patient to open his hand (C) to see the palmar pallor and then releasing the ulnar artery (D) or the radial artery (E) with the noticeable flushing.

EXAMINATION OF THE PRECORDIUM

The precordium is a term used to refer to the anterior aspect of the chest that overlies the heart.

INSPECTION

I. SHAPE OF THE CHEST (FIGURE 4 – 7)

The normal shape of the chest is bilaterally symmetrical and elliptical in cross section with a transverse diameter that is more than anteroposterior diameter in a ratio 7:5 and there is no bending to one side. Abnormal chest shape:

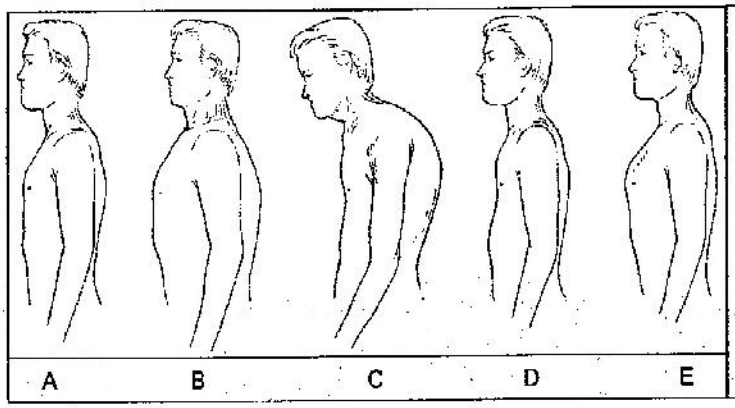


FIGURE 4 – 7. Normal chest shape (A), and abnormal shapes including barrel chest (B), Kyphosis (C), funnel chest (D), and pigeon chest (E).

1 Kyphosis (forward bending)	May alter the position of the apex beat and can lead to heart failure
2 Scoliosis (lateral bending)	May alter the position of the apex beat and can lead to heart failure. Kyphosis, scoliosis, or kyphoscoliosis may be idiopathic (80%), secondary to poliomyelitis, or associated with Marfan's syndrome.
3 Funnel chest (pectus excavatum)	This type of deformity (also called sternal depression) is a developmental defect involving a localised depression of the lower end of the sternum. Minor degree is common and harmless but with severe degree soft systolic murmur may be related
4 Pigeon chest (pectus carinatum)	It consists of a localized prominence of the sternum and adjacent costal cartilages (an outward bowing of the sternum and costal cartilages). This deformity occurs due to rickets, asthma, or with no obvious cause.
5 Barrel chest (emphysematous chest)	When the anteroposterior (AP) diameter is increased compared with the lateral diameter, the chest is described as barrel-shaped. An increase in the AP diameter compared with the transverse diameter (the thoracic ratio) beyond 0.9 is abnormal and is seen often in patients with severe asthma or emphysema. It is not always a reliable guide to the severity of the underlying lung disease and may be present in normal elderly people.

PRESENCE OR ABSENCE OF SCAR

During precordial examination the candidate should particularly look for three types of scars (FIGURE 4 – 8) which are related to the cardiovascular system examination. These are:

1 Median sternotomy (a cut down the middle of the sternum)	Most valve surgery requires cardiopulmonary bypass and for this it is very commonly used. It is occasionally hidden under a forest of chest hair. It is not specifically helpful, as it may also be a result of previous coronary artery bypass grafting.
2 left submammary scar (lateral thoracotomy scar)	This type of scar, which may be hidden under a pendulous breast, may indicate a previous closed mitral valvotomy. In this operation a stenosed mitral valve is opened through incision in the left atrial appendage; cardiopulmonary bypass is not required.
3 Infraclavicular scar	This type of scar reflects the placement of cardiac pacemaker or cardioverter-defibrillator box. These are usually under the right or left pectoral muscle just below the clavicle, are usually easily palpable and obviously metallic. The pacemaker leads may be palpable under the skin, leading from the top of the box. The box is normally mobile under the skin. Fixation of the skin to the box or stretching of the skin over the box may be an indication for repositioning. Erosion of the box through the skin is a serious complication because of the inevitable infection that will occur around this foreign body. Rarely a loose lead connection will lead to twitching of the muscles of the chest wall around the box. Penetration of the right ventricular lead into or through the right ventricular wall may lead to disconcerting paced diaphragmatic contractions (hiccups) at whatever rate the pacemaker is set. Defibrillator boxes are larger than pacemakers.

FIGURE 4 – 8. Scar of open heart surgery (A), mitral valvotomy (B), and pacemaker (C).

VISIBLE PULSATION

Visible pulsations in the chest could be normal cardiac impulse or pathologic finding (FIGURE 4 – 9).

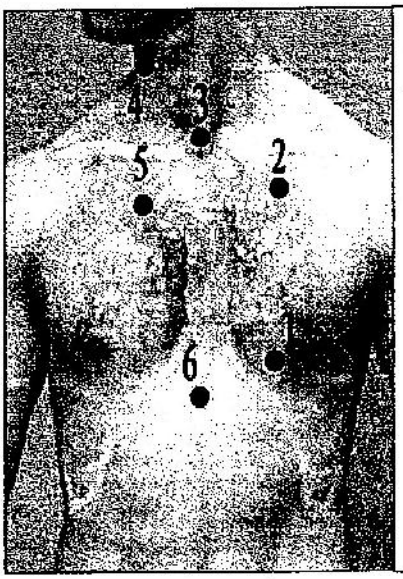


FIGURE 4 – 9. Visible precordial pulsation:

1. Apical pulsation can occur in left ventricular hypertrophy and in hypertrophic obstructive cardiomyopathy
2. Pulmonary area pulsation due to enlarged pulmonary artery
3. Suprasternal pulsation due to aortic arch aneurysm
4. Corrigan's sign which is brisk carotid pulsation that occurs in aortic regurgitation and in hyperdynamic circulation (pregnancy, exercise, thyrotoxicosis, anemia)
5. Right upper parasternal pulsation due to ascending aortic aneurysm
6. Pulsation at the epigastric area can normally occur in thin anxious person. It may occur due to abdominal aortic aneurysm, right ventricular hypertrophy, or due to pulsatile liver in tricuspid valve regurgitation.

PALPATION

LOCALIZATION OF APEX BEAT

Apex beat is defined as the lower most lateral most palpable gentle brief impulse that is located normally in the fifth left intercostal space at or medial to, the midclavicular line and has the capability to lift the finger overlying it. The cardiac impulse results from the heart rotating, moving forward and striking the chest wall during systole. Localization of apex beat should follow the following steps (FIGURE 4 – 10):

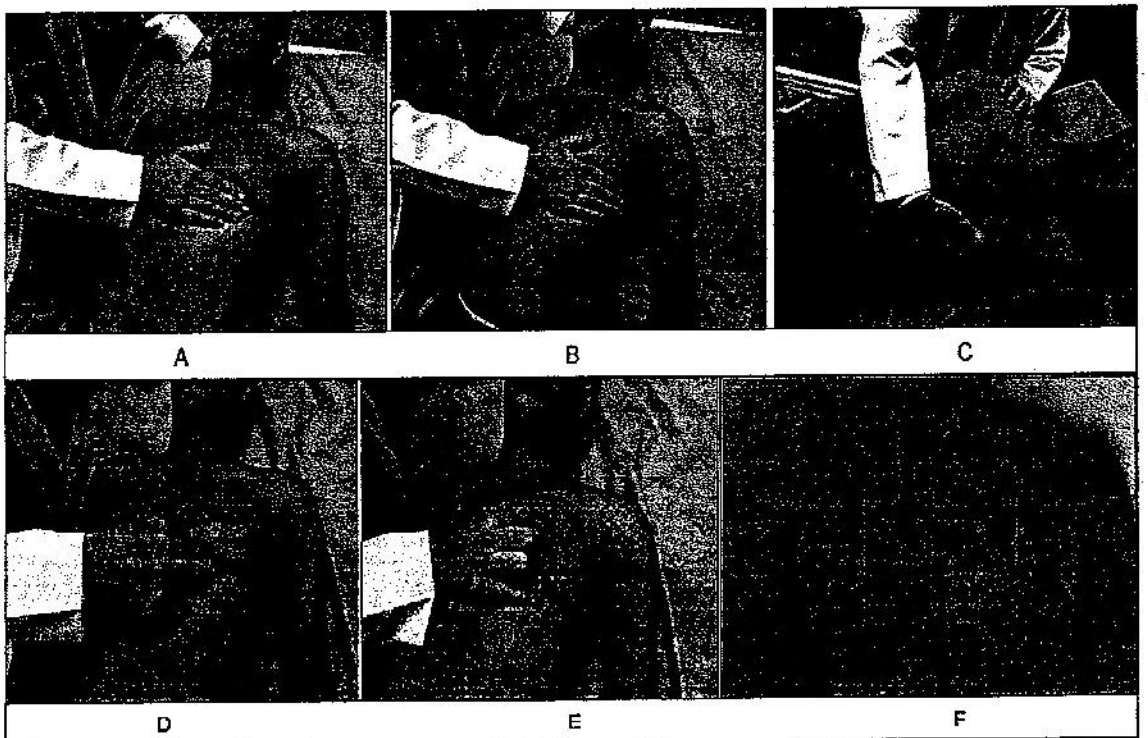


FIGURE 4 – 10. Localisation of the apex beat includes resting the patient in semi-recumbent position and then searching for the apex beat (A) which if not palpable in semi-recumbent position (B) one should turn the patient to the left lateral position (C). The apex beat is the impulse capable of slightly lifting your finger overlies it (D) and it is usually located in the fifth intercostal space (E) when the candidate counts downwards from the sternal angle. Coin is over the apex (F). Intercostal spaces are numbered. Vertical lines show right and left midclavicular lines. Care must be taken in identifying the left midclavicular line

1 Let the patient lie in a semi-recumbent posture (tilt the bed to 45°)
2 The right hand is firstly placed over and below the left nipple with the middle finger at about the fifth intercostal space and distal parts of fingers at the anterior axillary line
3 Wide area to be screened down wards and laterally so as to search for the lateral most and inferior most point at which the cardiac impulse can be palpated and this point can lift the finger overlying it with each systole.
4 Turn the patient to the left lateral decubitus if you can not detect the apex beat.
5 The palpable area should be finely adjusted by the tip of the middle finger. After fine adjustment this area is called the apex beat
6 Sternal angle corresponds to the second rib so count from second intercostal space to the apex beat to determine at which intercostal space the apex beat is lying. At the same time imagine a line from left mid clavicle down wards to determine whether the apex beat is displaced (BOX 4 – 1) to one side or not. Note that the apex beat is palpable in only about 50% of adults (BOX 4 – 1).

CLINICAL CLUE

Be sure you are not feeling your finger pulsation. If so detect your right radial pulse with your left hand.

BOX 4 – 1. Causes of absent apex beat and displaced apex beat

Medial displacement	Lateral displacement	Absent apex beat	
Collapsed right lung	Left ventricular dilatation	Obesity	Pericardial effusion
Fibrosed right lung	Chest wall deformities	Muscular patients	Pericardial constriction
	Right pneumothorax	Emphysema	Dextrocardia
	Right pleural effusion	Right pneumonectomy	

CHARACTER OF THE APEX BEAT

Normal character of the apex beat is defined above. The candidate should assess the character of apex beat with his/her finger tips which may take one of the following forms:

1	The pressure loaded (heaving) apex beat	It is a forceful, sustained, and undisplaced impulse. This occurs with aortic stenosis or hypertension
2	The volume loaded (thrusting) apex	It is a displaced, diffuse, non-sustained impulse. This occurs most commonly in advanced mitral regurgitation or aortic regurgitation
3	The dyskinetic apex beat	It is an uncoordinated impulse felt over a larger area than normal in the precordium and is usually due to left ventricular dysfunction
4	Double apex impulse	Two distinct impulses are felt with each systole. This impulse is characteristic of hypertrophic cardiomyopathy
5	Tapping apex beat	It is non displaced palpable impulse that occurs in mitral stenosis

HEAVES (LIFTS), THRILLS, AND PALPABLE SOUNDS

Heave is a palpable impulse that lifts your hand noticeably. It is of two types; left parasternal heave due to right ventricular hypertrophy (due to pulmonary hypertension) or apical heave due to left ventricular hypertrophy (due to hypertension or aortic stenosis). Heave or lift is best detected with the ball of the hand (FIGURE 4 – 11). Thrill is the superficial vibratory sensations felt on the skin overlying an area of turbulence and feels similar to a mobile telephone vibrating. The presence of a thrill indicates a loud murmur. Thrills are best felt with the metacarpal bones rather than the fingertips by applying very gentle pressure on the skin (FIGURE 4 – 11). Remember that any murmur may produce thrills. Apical thrills of mitral stenosis can be more easily felt with the patient rolled over to the left side (the left lateral position) as this brings the apex closer to the chest wall. Thrills may also be palpable over the base of the heart. These may be maximal over the pulmonary or aortic areas, depending on the underlying cause, and are best felt with the patient sitting up, leaning forwards and in full expiration. In this position the base of the heart is moved closer to the chest wall. A thrill that coincides in time with the apex beat is called a systolic thrill; one that does not coincide with the apex beat is called a diastolic thrill. The heart sounds are not palpable in health. Palpable first heart sound (S1) in the mitral area occurs in mitral stenosis; palpable (S2) in the pulmonary area (P2) occurs in pulmonary hypertension; palpable third heart sound (S3) in the mitral area suggests constrictive pericarditis (pericardial knock); palpable fourth heart sound (S4) in the mitral area suggests hypertrophic obstructive cardiomyopathy. Prior to performing palpation the candidate should know the precordial areas as shown in FIGURE 4 – 11.

The technique of examination should follow (FIGURE 4 – 12) the following steps:

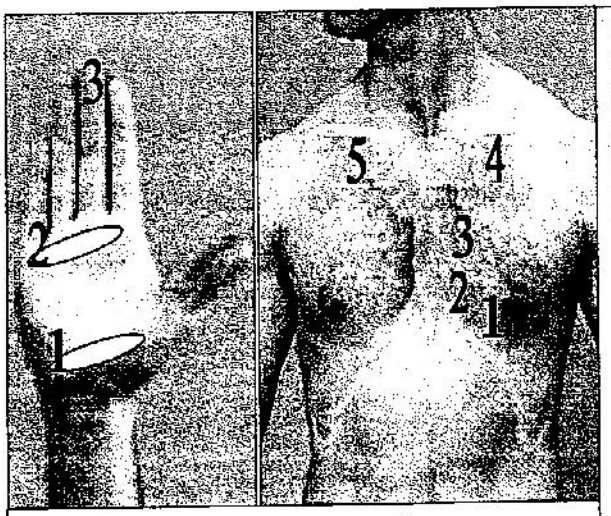


FIGURE 4 – 11. Figure A shows the sites in the hand suitable for palpation including that for heart sounds (1), for thrills (2), and for heave (3). Figure B shows the sites of the precordial areas including the mitral area (1), the tricuspid area or the left parasternal area (2), the Erb's area (3) the pulmonary area (4), and the aortic area (5).

FIGURE 4 – 12. Detection of palpable heart sounds, heaves, and lifts. After apex beat localization, the candidate should search for palpable heart sounds (A), thrills (B), and heave (lifts) (C) at the mitral area. Left parasternal heave (D) and lifts (E) should next be looked for at the left parasternal area. The candidate after that should search for VSD thrills at the Erb's area (F). Palpable second heart sound (P2) should be looked for at the pulmonary area (G) and then looking for thrills (H). The candidate then looks for thrills at the aortic area (I).

J K L

FIGURE 4 – 12 continued. The candidate then asks the patient to turn to the left lateral position (J) to look for diastolic thrills of mitral valve stenosis. Finally the candidate asks the patient to sit and check for systolic thrills of aortic stenosis and pulmonary stenosis provided that the patient is asked to fully expire breath.

PERFORM THE FOLLOWING STEPS WHILE YOUR LEFT THUMB OVER THE RIGHT CAROTID TO DETECT WHETHER THE THRILL IS SYTOLIC OR DIASTOLIC

1 STEP I At the mitral area check for
Palpable S1, S3, and S4
Thrills and heave (lift)

2 STEP II At the left parasternal area check for
Thrills and heave (lift)

3 STEP III At the Erb's area check for
Thrills only

4 STEP IV At the pulmonary area check for
Palpable P2
Thrills

5 STEP V At the aortic area check for
Thrills only

6 STEP VI Turn the patient to the left side and check for diastolic thrill of mitral stenosis

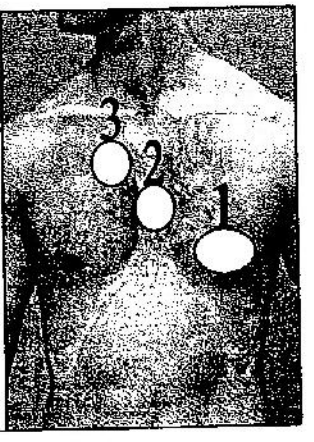
7 STEP VII Ask the patient to stand and check for systolic thrills of aortic stenosis and pulmonary stenosis provided that the patient is asked to fully expire breath

PERCUSSION

Percussion is not usually performed to the precordium. The candidate is sometimes asked about the clinical benefits of percussion. This is shown in FIGURE 4 – 13.

It is possible to define the cardiac outline by means of percussion but this is NOT ROUTINE. Percussion is most accurate when performed in the fifth intercostal space. The patient should lie supine and the examiner percusses from the anterior axillary line towards the sternum. The point at which the percussion note becomes dull represents the left heart border. A distance of more than 10.5 cm between the border of the heart and the middle of the sternum indicates cardiomegaly. The sign is not useful in the presence of lung disease.

FIGURE 4 – 13. The sites of percussion:
1. Reduced or absent cardiac dullness in case of emphysema
2. Area of abnormal dullness in case of pericardial effusion
3. Area of abnormal dullness in case of large ascending aortic aneurysm



AUSCULTATION

The earpieces of the stethoscope are directed anteriorly or parallel to the direction of the external auditory canal. If the earpieces are put in backward, the openings of the earpieces will impinge on the wall of the external canal and lower the intensity of the sounds. The earpieces should fit properly so as to be comfortable but tight enough to exclude

external noises. The bell of the stethoscope should be applied lightly to the skin, whereas the diaphragm should be pressed tightly to the skin. High-pitched sounds, such as first heart sound (S1), second heart sound (S2), all systolic murmurs, and all diastolic murmurs (except mitral stenosis) and opening snaps, are better heard with the diaphragm. Low pitched sounds, such as third heart sound (S3), fourth heart sound (S4), and mitral diastolic murmur, are better heard with the bell. The candidate should be on the right side of the patient while the patient lies in a semi-recumbent posture. The candidate should auscultate the precordial areas according to the Z – rule (FIGURE 4 – 14). Proper auscultation requires a quiet area and every attempt should be made to eliminate extraneous noises. Never listen through any type of clothing.

The technique of auscultation involves the following steps (FIGURE 4 – 15):

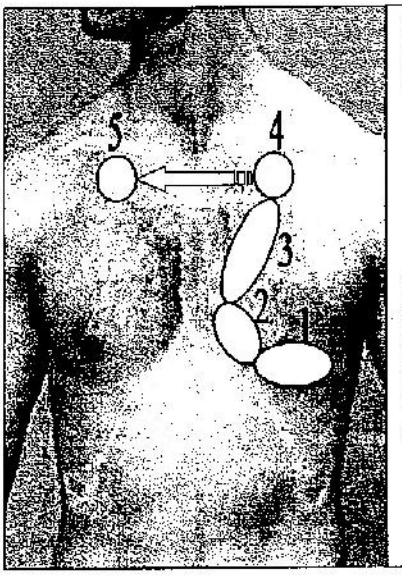


FIGURE 4 – 14. The precordial areas include:
1. Mitral areas
2. Left parasternal area
3. Erb's area
4. Pulmonary area
5. Aortic area

The candidate should listen over these areas starting from the mitral area and ending in the aortic area in a Z-shape cycle.

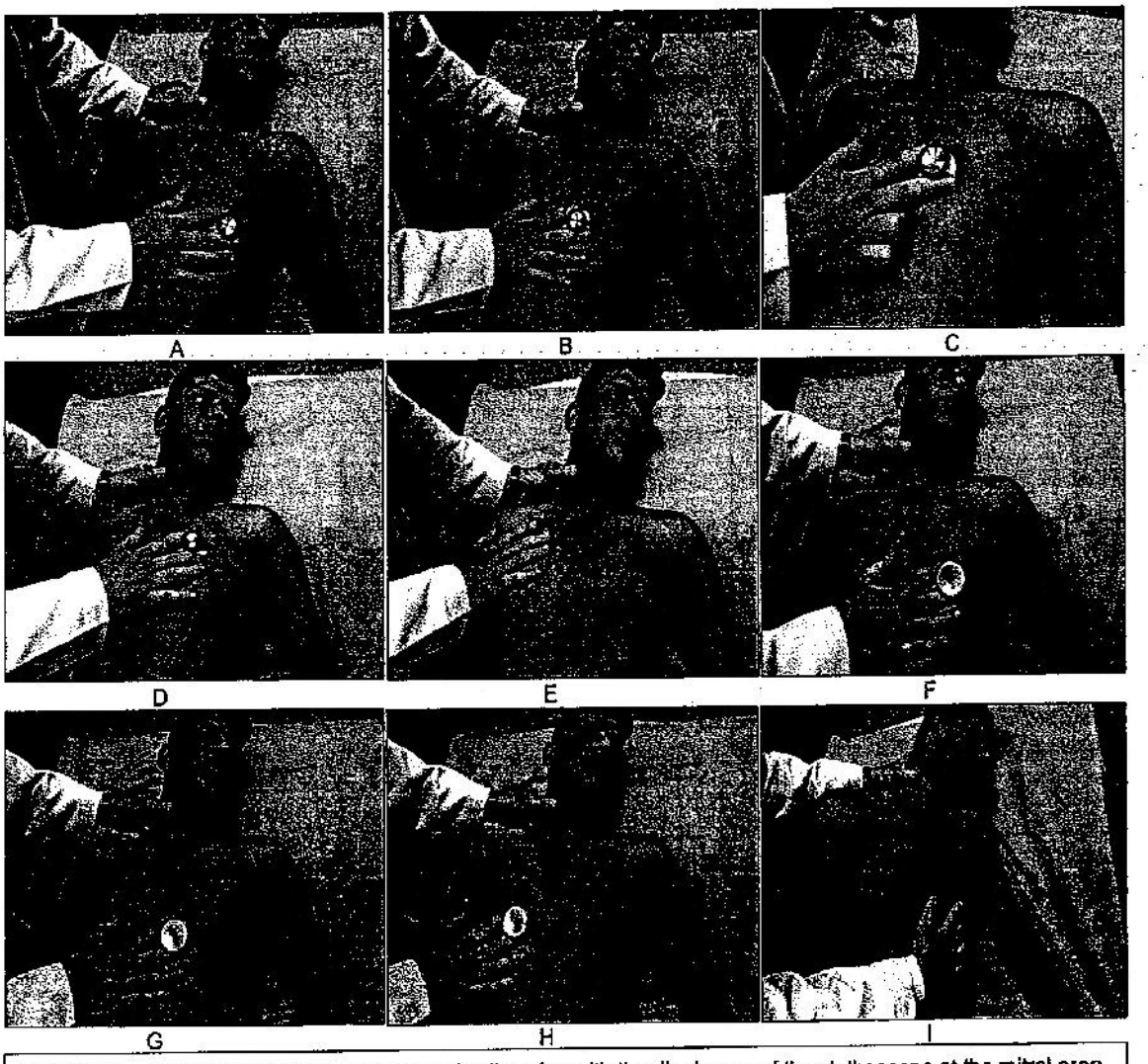
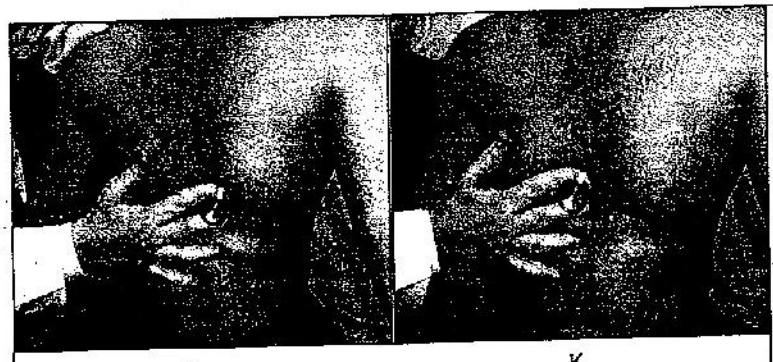


FIGURE 4 – 15. Precordial auscultation includes listening with the diaphragm of the stethoscope at the mitral area (A), left parasternal area (B), Erb's area (C), pulmonary area (D), and the aortic area (E). The mitral area is then ascultated with the bell of the stethoscope (F) for left ventricular third and fourth heart sounds. The candidate then listen over the left parasternal (G) and right parasternal areas (H) for right ventricular third and fourth heart sounds. The patient is then asked to turn to the left lateral position (I) to listen with the bell for mitral stenosis.



J K

FIGURE 4 – 15 continued. Finally the patient is asked to sit up and lean forward. The patient is asked to exhale and hold the breath while the candidate, using the diaphragm, listens for the high pitched diastolic murmur of aortic regurgitation at the left parasternal (J) and right parasternal areas (K).

1 All precordial areas are examined while the patient is in a semi-recumbent posture and the candidate's left thumb over the carotid artery to determine the sound whether systolic or diastolic. Systolic sound is that which coincides with the carotid pulsation and with S1; the diastolic sound is that which comes after the carotid pulse and after S1
2 Using a systematic approach, the candidate starts at the apex and carefully listens to the heart sounds and added sounds with the diaphragm of the stethoscope according to the Z-rule
3 The candidate then applies the bell to mitral area to listen for left ventricular third and fourth heart sounds and to the left/right parasternal area for right ventricular third and fourth heart sounds
4 The patient is then instructed to turn onto the left lateral position. The candidate should listen at the apex for the low-pitched diastolic murmur of mitral stenosis, which is best heard with the bell
5 Finally, the patient is asked to sit up, lean forward, and to exhale and hold the breath while the candidate, using the diaphragm, listens for the diastolic murmur of aortic regurgitation at the right and left parasternal areas

During auscultation, the candidate should determine the first heart sound (normal, loud, or diminished), the second heart sound (normal, loud, diminished, or split), and added sounds (third heart sound, fourth heart sound, opening snaps, ejection clicks, mid-systolic click, prosthetic valve sounds, pericardial rub, and murmur).

BOX 4 – 2. Heart sound findings	
First heart sound	
Loud	Soft
Mitral stenosis Tachycardia Short PR interval (WPW syndrome) Increased cardiac output	Mitral regurgitation Long PR interval (first degree heart block) Low cardiac output
Second heart sound	
Loud	Physiologic splitting (A2 before P2)
Systemic hypertension (aortic component) Pulmonary hypertension (pulmonary component)	The splitting increases with inspiration and decreases with expiration. It occurs in pregnancy and children
Soft	Fixed splitting
Low cardiac output Aortic stenosis	Splits in inspiration and expiration and occurs in atrial septal defect (ASD) and in arrhythmogenic right ventricular dysplasia
Enhanced physiologic splitting (A2 before P2)	Reversed (paradoxical) splitting (P2 before A2)
The width of splitting markedly increases with inspiration but decreases with expiration because the defect is in the right side that delays P2 component of S2. Thus A2 before P2 , occurs in • Dilated right ventricle • Pulmonary stenosis and hypertension • RBBB	The sound splits in expiration and comes together during inspiration because of left side defect that delays A2 component of S2 and thus P2 component comes before A2. It occurs in • Hypertrophic obstructive cardiomyopathy (HOCM) • Aortic stenosis • LBBB

FIRST HEART SOUND (S1)
It is the sound of mitral and tricuspid valves closure. Place the stethoscope over the mitral area and your left thumb over the patient's right carotid artery; you will hear the sound of the heart as lub-dub and the carotid wave will be felt. S1 is lub in lub–dub and is the sound that immediately precedes the carotid pulse wave.

SECOND HEART SOUND (S2 =A2 + P2)

It is the sound of aortic and pulmonary valves closure. The components of second heart sound are thus pulmonary and aortic components. Place the stethoscope over the pulmonary area and your left thumb over the patient's right carotid artery, you will hear the sound of the heart as lub-dub and the carotid wave will be felt. S2 is dub in lub-dub and is the sound that immediately follows the carotid pulse wave (BOX 4 – 2).

THIRD HEART SOUND (S3)

It is the sound of rapid filling of ventricles during early diastole best heard at the apex with the stethoscope bell press lightly over mitral area (BOX 4 – 3). When third heart sound is accompanied by tachycardia the rhythm is called triple or gallop rhythm that is similar to the sound produced by galloping horse. S3 is heard after S2 as lub-dub–dum. A left ventricular S3 is louder at the apex than at the left sternal edge, and is louder on expiration. A right ventricular S3 is louder at the left sternal edge and with inspiration. It occurs in right ventricular failure or constrictive pericarditis. In pericardial constriction the sudden rush of blood into the ventricle during diastole causes a very loud third heart sound which is often termed pericardial knock.

FOURTH HEART SOUND (S4)

It is the sound that is produced due to forceful atrial contraction during late diastole best heard at the apex with the stethoscope bell press lightly over mitral area (BOX 4 – 3). When fourth heart sound is accompanied by tachycardia the rhythm is called triple or gallop rhythm. Fourth heart sound (S4) is heard just before first heart sound (S1) as da-lub-dub. It does not occur if the patient is in atrial fibrillation, because the sound depends on effective atrial contraction, which is lost when the atria fibrillate. The left ventricular S4 is louder at the apex while the right ventricular S4 is louder at the left or right parasternal area and occurs in pulmonary hypertension or pulmonary stenosis.

CLINICAL CLUE

When both the third and fourth heart sounds are heard with tachycardia, the expression used is called summation gallop and the rhythm is called quadruple rhythm.

BOX 4 – 3. Causes of third heart sounds and fourth hearth sounds

Third heart sound		Fourth heart sound	
Physiological	Pathological	Physiological	Pathological
Healthy young adults Athletes Pregnancy	Heart failure Mitral regurgitation Tricuspid regurgitation Constrictive pericarditis Cardiac tamponade Restrictive cardiomyopathy	Healthy trained athletes children	HOCM Pressure over load (systemic hypertension and aortic stenosis) Ischemic heart disease

OPENING SNAP

It results from the restricted opening of a stenosed mitral valve (rarely tricuspid valve). It is heard after S2 in mitral stenosis at the mitral area by using a diaphragm. It is only audible when the valve is mobile. When the left atrial pressure is high, the opining snap closely follows S2. Conversely, when the left atrial pressure is relatively normal the snap occurs later.

EJECTION CLICKS

It results from the restricted opening of a stenosed aortic or pulmonary valve. It is heard after S1 in aortic stenosis at the aortic area and in pulmonary stenosis at the pulmonary area. It indicates that the lesion is valvular and is only audible when the valve is mobile. Thus it disappears once the valve becomes calcified.

HOW CAN YOU DIFFERENTIATE BETWEEN EJECTION CLICK AND FOURTH HEART SOUND?

The fourth heart sound is not heard when pressure is applied on the chest with the piece of the stethoscope, but pressure does not eliminate the ejection sound

MID-SYSTOLIC CLICK

It occurs in mitral valve prolapse.

PROSTHETIC VALVE SOUNDS

Mechanical heart valve produces a quiet opening click and a loud metallic closing sound commonly palpable and often audible without a stethoscope. Two types of valve prosthesis:

1 Mitral valve prosthesis S1 (loud) + S2 + click (quiet)

2 Aortic valve prosthesis S1 + click (quiet) + S2 (loud)

PERICARDIAL RUB

It is a superficial scratching sound likes a friction between two rough areas similar to that of pleural rub. It is generally best heard at the left of the lower sternum with the patient leaning forward and breathing out using the diaphragm of the stethoscope. This sound has diastolic and systolic components. Some causes of pericarditis and therefore pericardial rub are shown in BOX 4 – 4.

BOX 4 – 4. Some causes of pericarditis

Acute myocardial infarction	Rheumatic fever
Post myocardial infarction (Dressler's syndrome)	Myxedema
Viral (e.g. Coxsackie A and B)	Familial Mediterranean fever
Bacterial infection (pneumococcus, staphylococcus)	Drug-induced (e.g., hydralazine, phenytoin, isoniazide)
Tuberculosis	Postcardiac injury
Uremia	Post irradiation
Malignant disease (primary and secondary)	
Trauma (e.g. blunt chest injury)	
Connective tissue disease (e.g. SLE, rheumatoid arthritis)	

MURMURS

Heart murmurs are produced by turbulent blood flow in the heart. They are either produced by abnormal valve function or arise from an increased volume, or velocity of blood flowing through a normal valve. The sequence of precordial auscultation is described earlier. However, once cardiac murmur is detected the candidate needs to fully describe that murmur in the following ways:

1 Area of maximum intensity (FIGURE 1 – 16)

This means the area at which the cardiac murmur is louder. Each murmur has an area of maximum intensity

2 Timing of the murmur (BOX 4 – 5)

Listen to the murmur and palpate the carotid artery:
The murmur is systolic if the sound comes with the carotid wave. If so, the murmur is ejection if it comes in the early time of S1; pansystolic when occurs along the whole S1; late systolic when occurs in the late time of S1.
The murmur is diastolic if the sound comes after the carotid wave.
The murmur is continuous if the sound comes along the carotid wave

3 Character of the murmur

Any murmur may have musical quality. All ejection systolic murmurs have harsh quality; regurgitant murmurs have blowing quality; mitral stenosis has rumbling quality; continuous murmur due to PDA has machinery quality. Sea gull quality may be due to mitral regurgitation with rupture of chordae tendineae or due to aortic stenosis.

4 Radiation of the murmur

Murmurs radiate in the direction of the blood flow to specific sites outside the precordium. The pansystolic murmur of mitral regurgitation radiates towards the left axilla, the murmur of ventricular septal defect towards the right sternal edge, and that of aortic stenosis to the carotid arteries.

5 Grading of the murmur (BOX 4 – 6)

Systolic murmurs are graded according to their severity into six grades while diastolic murmurs are graded into four grades. The record of the grades should be stated for example 5/6 i.e. fifth out of six grades

6 Relation to respiration (this task is belonging to the postgraduate doctors)

Postgraduate students should determine whether the added sound increases or decreases with inspiration or expiration. Generally speaking all right heart added sounds including murmurs increase with inspiration except ejection click of pulmonary valve stenosis. All left sided heart sound including murmurs increase with expiration

7 Relation to certain maneuvers (this task is belonging to the postgraduate doctors)

With Valsalva maneuver or with standing from squatting most murmurs decrease in intensity except HCM and MVP.
With squatting from standing, with passive leg rising and with hand grip most murmur become louder in intensity except HCM and MVP

8 Provisional diagnosis

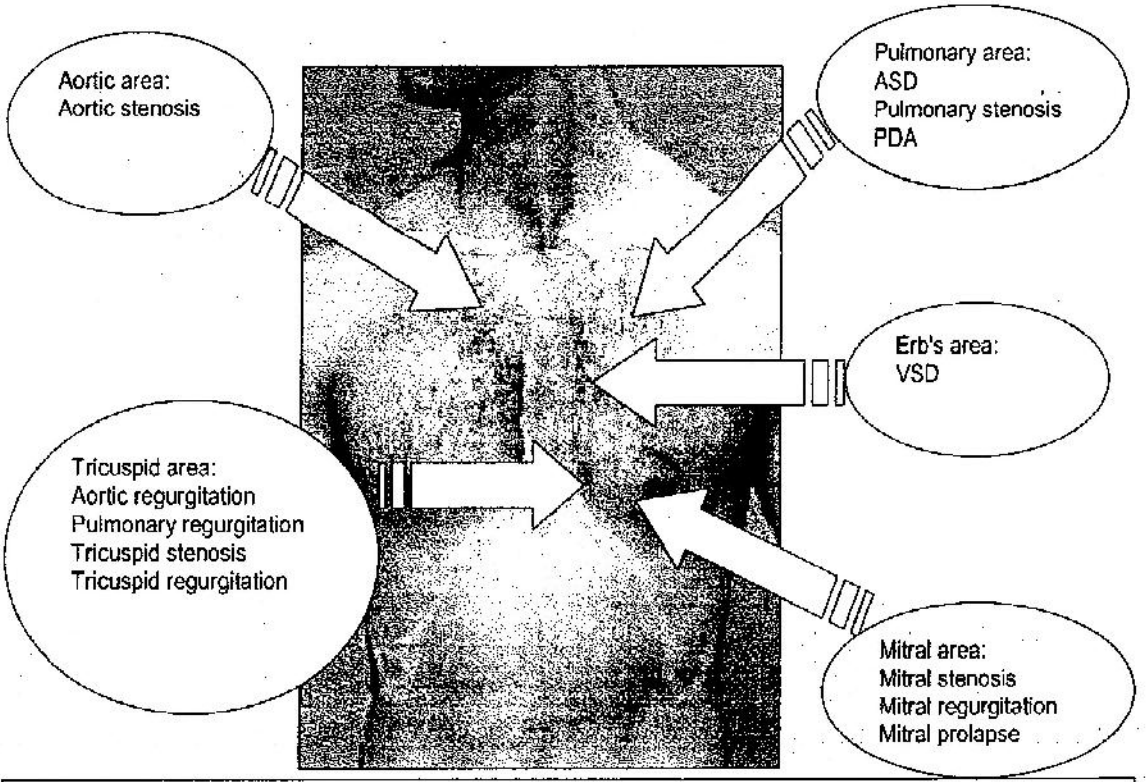


FIGURE 4 – 16. The areas of maximum intensity of cardiac murmurs.

BOX 4 – 5. Timing of cardiac murmurs

Systolic murmurs	Diastolic murmurs	Continuous murmurs
Ejection systolic murmurs	Early diastolic murmurs	Patent ductus arteriosus Venous hum Coarctation of aorta Arteriovenous fistula (coronary artery, pulmonary, systemic) Aortopulmonary connection (e.g. congenital, Blalock shunt) Rupture of sinus of Valsalva into right ventricle or atrium 'Mammary souffle' (in late pregnancy or early postpartum period)
• Aortic stenosis • Pulmonary stenosis • Atrial septal defect (ASD)	• Aortic regurgitation • Pulmonary regurgitation	
Pansystolic murmurs	Mid-diastolic murmurs	
• Mitral regurgitation • Tricuspid regurgitation • Ventricular septal defect (VSD)	• Mitral stenosis • Tricuspid stenosis	
Late systolic murmurs	Austin flint murmur	
• HOCM • Mitral valve prolapse		

FOR POSTGRADUATE DOCTORS

The systolic murmur of hypertrophic cardiomyopathy is caused by turbulent flow and obstruction in the left ventricular outflow tract from the thickened interventricular septum. In severe cases, systolic anterior motion of the mitral valve apparatus into the left ventricular outflow tract contributes to the systolic murmur. If mitral valve leaflet coaptation is affected, there may be concurrent mitral regurgitation. The stand-to-squat maneuver and passive leg lift transiently increase venous return (preload), which increases left ventricular chamber size and volume. As a consequence, there is less relative obstruction and turbulence in the left ventricular outflow tract, decreasing murmur intensity. The Valsalva maneuver and the squat-to-stand maneuver transiently decrease venous return, with the septum and anterior mitral leaflet brought closer together. Turbulent flow—and the murmur—are increased. Handgrip exercise increases after load and decreases the pressure gradient across the left ventricular outflow tract, so murmur intensity for hypertrophic cardiomyopathy is decreased.

In a patient with mitral valve prolapse, the Valsalva maneuver moves the click murmur complex earlier in systole and may increase the intensity of the murmur. Similarly, decreased preload (smaller left ventricular chamber size) increases systolic buckling of the valve leaflets and the murmur lengthens or intensifies. The carotid upstroke is normal in mitral valve prolapse.

BOX 4 – 6. Grading of a murmur	
Grade 1	Very soft, heard by an expert in optimal conditions
Grade 2	Soft, heard by a non-expert in optimal conditions
Grade 3	Moderate; no thrill
Grade 4	Loud with associated thrill
Grade 5	Very loud, heard over a wide area
Grade 6	Extremely loud, heard with-out stethoscope

AORTIC STENOSIS

Mild or moderate aortic stenosis is usually a symptomatic, but severe aortic stenosis causes exertional dyspnoea, exertional syncope or even sudden death. Clinical features are provided in BOX 4 – 7. In some cases, the murmur may disappear over the sternum and reappear over the LV apex, thereby giving the false impression that a murmur of mitral regurgitation is also present (Gallivardan's phenomenon)

BOX 4 – 7. Clinical features of aortic stenosis	
Symptoms (mnemonic: 5 As)	Signs (mnemonic 5 Ss)
A symptomatic Arrhythmia Angina Attack of hypotension, syncope, or sudden death Asthma (cardiac asthma)	Systolic murmur, thrill, and click S abnormalities (S2 abnormality in the form reversed splitting, S3 and/or S4) Systolic blood pressure is near to diastolic blood pressure (narrow pulse pressure) Slow and rising pulse (anacrotic pulse) Sustained and forceful apex beat

The three principle causes of aortic valve stenosis include senile degenerative aortic stenosis, calcification of bicuspid valve, and rheumatic aortic stenosis.

PULMONARY VALVE STENOSIS

This can occur in the carcinoid syndrome but is usually congenital, when it may be isolated or associated with other abnormalities such as Fallot's tetralogy. Pulmonary stenosis may give rise to certain features suggestive of severity (BOX 4 – 8).

BOX 4 – 8. Signs of severe pulmonary stenosis
Loud harsh murmur
Inaudible pulmonary closure sound (P2)
Increased right ventricular heave
Prominent a waves in the jugular pulse
ECG evidence of right ventricular hypertrophy
Post-stenotic dilatation in the pulmonary artery on the chest X-ray

ATRIAL SEPTAL DEFECT (ASD)

ASD is one of the congenital heart disease in which most children are free of symptoms for many years or complain from dyspnoea, chest infection, cardiac failure and arrhythmias especially atrial fibrillation. Functional mid-diastolic flow murmur at the left parasternal area may also be present.

MITRAL REGURGITATION

Rheumatic disease is the principal cause for mitral regurgitation (BOX 4 – 9). Chronic mitral regurgitation causes few symptoms as dyspnoea and pulmonary edema, but acute mitral regurgitation causes marked symptomatic deterioration. In general, the patient complains from dyspnoea, fatigue, palpitation as well as edema and ascites due to right heart failure. The murmur is radiated into the back (when anterior leaflet is abnormal), axilla, or anterior chest wall across precordium mimicking aortic stenosis (when posterior leaflet is abnormal).

BOX 4 – 9. Causes of mitral regurgitation	
Rheumatic valve disease Mitral valve prolapse Dilatation of the left ventricle and mitral valve ring (e.g. coronary artery disease, cardiomyopathy)	Damage to valve cusps and chordae (e.g. rheumatic heart disease, endocarditis) Papillary muscle ischemia or infarction Inferior myocardial infarction

TRICUSPID REGURITATION

The most frequent cause is functional as a result of right ventricular dilatation. Symptoms are usually non specific but may include raised JVP, and pulsatile liver.

VENTRICULAR SEPTAL DEFECT (VSD)

Congenital VSD occurs as a result of incomplete septation of the ventricles, but acquired VSD occurs as complication of acute myocardial infarction. VSD may be a part of tetralogy of Fallot (pulmonary stenosis, overriding of aorta, VSD and right ventricular hypertrophy). VSD causes heart failure in infantile period.

HYPERTROPHIC CARDIOMYOPATHY (HCM)

In this condition the muscle fibres of the ventricles become inappropriately hypertrophied and distorted. Hypertrophy may be generalised or confined largely to the interventricular septum (asymmetric septal hypertrophy) or other regions (e.g. apical). When interventricular septum contracts, it bulges into outflow of left ventricle causing some degree of obstruction which produces ejection systolic murmur mimicking aortic stenosis but the distinguishing feature is typically jerky carotid arterial pulse. In addition, when papillary muscles involved there is mitral regurgitation.

MITRAL VALVE PROLAPSE

Mitral valve prolapse (MVP), also variously termed the systolic click-murmur syndrome, Barlow's syndrome, floppy-valve syndrome, and billowing mitral leaflet syndrome, is a relatively common. It is caused by congenital anomalies or degenerative myxomatous changes and is sometimes a feature of connective tissue disorders such as Marfan's syndrome. The most important finding is the mid- or late (non ejection) systolic click. Systolic clicks may be multiple and may be followed by a high pitched, late systolic murmur, which occasionally is whooping or honking and is heard best at the apex.

AORTIC REGURGITATION

Aortic regurgitation may be caused by primary valve disease or by primary aortic root disease. In chronic aortic regurgitation, the high stroke volume and reduced systemic arterial resistance result in a wide pulse pressure, which may generate a number of signs, including a rapidly rising pulse (water-hammer), Corrigan's pulse (sharp upstroke and rapid decline of the carotid pulse), de Musset's sign (head bobbing), a booming (pistol-shot) sound can be heard over the femoral arteries (Traube's sign), and a to-and-fro murmur (Duroziez's sign) is audible if the femoral artery is lightly compressed with a stethoscope, and Quincke's pulse (systolic plethora and diastolic blanching in the nail bed when gentle traction is placed on the nail). Perhaps the most reliable of physical signs indicating severe aortic regurgitation is Hill's sign, an increase in femoral systolic pressure of 40 mm Hg or more when compared with systolic pressure in the brachial artery.

AUSTIN FLINT MURMUR

It is seen in severe aortic incompetence due to partial closure of the anterior mitral valve cusps caused by the regurgitant streams.

PULMONARY REGURGITATION (GRAHAM STEEL MURMUR)

It may be secondary to pulmonary artery dilatation in pulmonary hypertension producing a murmur similar in quality and site to that of aortic regurgitation, but here there are signs of pulmonary hypertension (BOX 4 – 10).

BOX 4 – 10. Signs of pulmonary hypertension
Elevation of the JVP (with a prominent 'a' wave if in sinus rhythm)
A parasternal heave (right ventricular hypertrophy)
Accentuation of the pulmonary component of the second heart sound
Early diastolic murmur due to pulmonary regurgitation.

MITRAL STENOSIS

Mitral stenosis is almost always rheumatic in origin. Normally the mitral orifice is 5 cm^2 in diastole. Patient usually remain a symptomatic even the orifice became 2 cm^2 (moderately severe), but when the orifice is 1 cm^2 (severe stenosis), the symptoms will develop. The first heart sound is usually accentuated and slightly delayed. The pulmonic component of the second heart sound also is often accentuated, and the two components of the second heart sound are closely split. The opening snap (OS) of the mitral valve is most readily audible in expiration at, or just medial to the cardiac apex. The OS is followed by a low-pitched, rumbling, diastolic murmur, heard best at the apex with the patient in the left lateral recumbent position. It is accentuated by mild exercise (e.g., a few rapid sit-ups) carried out just before auscultation. In patients with sinus rhythm, the murmur often reappears or becomes louder during atrial systole (presystolic accentuation).

TRICUSPID STENOSIS

It is similar to mitral stenosis, but it is rare.

CONTINUOUS MURMURS

A patent ductus arteriosus causes a continuous murmur as long as the pressure in the pulmonary artery is much below that in the aorta. When pulmonary hypertension is present, the diastolic portion may disappear, leaving the murmur confined to systole. The innocent cervical venous hum is a continuous murmur usually audible over the medial aspect of the right supraclavicular fossa with the patient upright. This condition is usually due to kinking and partial obstruction of one of the larger veins in the neck. The hum is usually louder during diastole and can be abolished instantaneously by digital compression of the ipsilateral internal jugular vein. Transmission of a loud venous hum to the area below the clavicles may result in a mistaken diagnosis of patent ductus arteriosus (BOX 4 – 11).

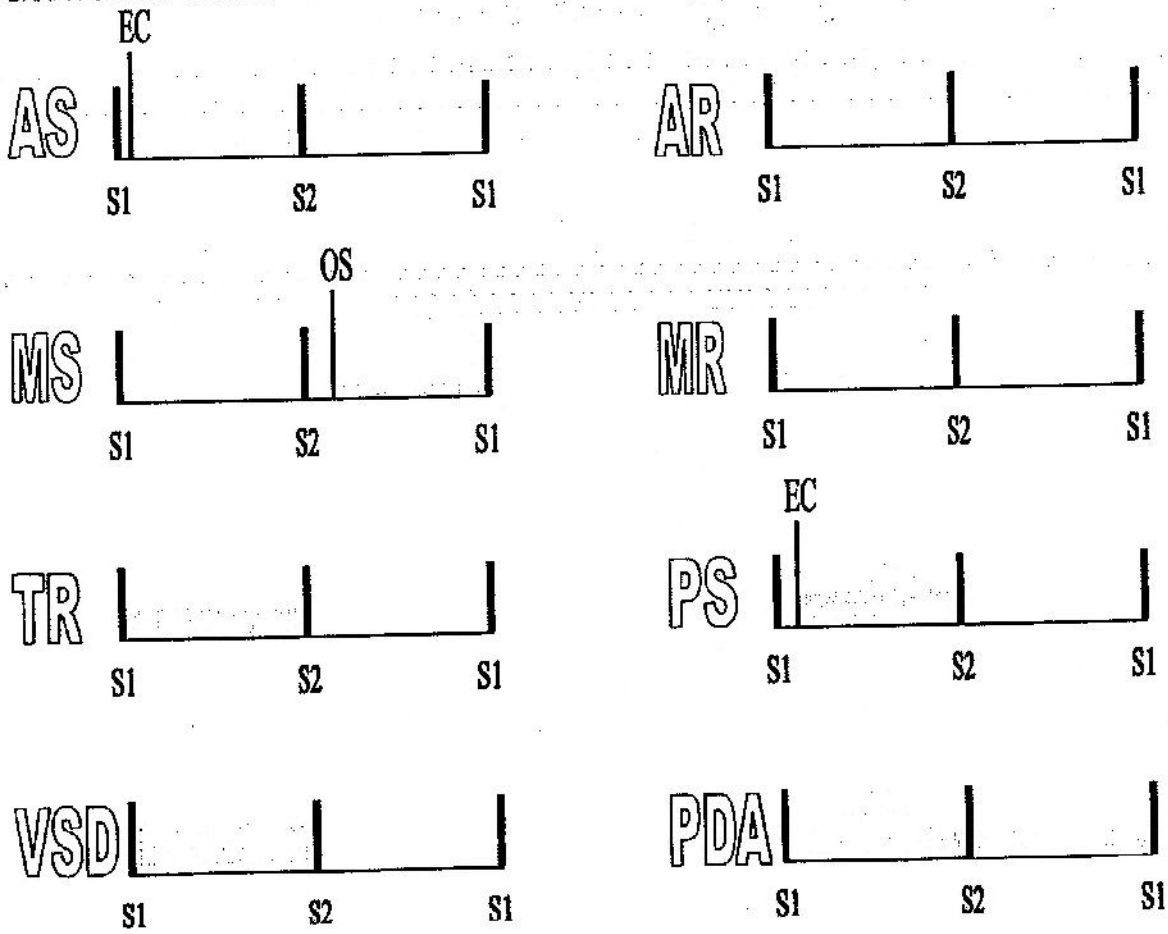
BOX 4 – 11. Distinction between PDA and venous hum
The venous hum can be obliterated by pressure on the neck which provides a complete obstruction of the vein
The venous hum can be obliterated by altering the position of the neck so as to relieve the venous obstruction

BENIGN (INNOCENT) MURMUR

Benign murmurs do not occur in diastole (BOX 4 – 12) and systolic murmurs that radiate or are associated with a thrill are almost always pathological.

BOX 4 – 12. The features of benign or innocent	
Soft	Not accompanied by thrill
Mid systolic	No radiation
Heard at the left sternal edge	No other cardiac abnormalities

GRAPHICAL REPRESENTATION OF THE CARDIAC MURMURS



EXAMPLES

Examination of the precordium had shown that the patient had normal chest shape, no obvious scar, but there is limited area of epigastric pulsation. The apex beat was localised to the fifth intercostal space slightly medial to left midclavicular line, of normal character gentle brief impulse, and there were no palpable heart sounds, thrills, or lifts. There was normal first heart sound, normal second heart sound, and no added sounds.

What is the interpretation?
The interpretation is normal precordial examination

Examination of the precordium had shown that the patient had normal chest shape, no obvious scar, but there is limited area of epigastric pulsation. The apex beat was displaced to the sixth intercostal space at the left anterior axillary line, diffused and non-sustained, and there were no palpable heart sounds, thrills, or lifts. There was soft first heart sound, normal second heart sound, and systolic murmur. The murmur was pansystolic murmur, maximally localised at the mitral area, of blowing quality, third grade out of sixth, and radiated into the left axilla.

What is the interpretation?
The interpretation is mitral valve regurgitation

Examination of the precordium had shown that the patient had normal chest shape, no obvious scar, but there is limited area of epigastric pulsation with raised JVP. The apex beat was localised to the fifth intercostal space at the left midclavicular line, forceful sustained and non- displaced, and there was no palpable heart sounds, systolic thrill at the aortic area, with no lifts. There was normal first heart sound, soft with paradoxical splitting of second heart sound, and systolic murmur. The murmur was ejection systolic murmur, maximally localised at the aortic area, of rasping quality, fourth grade out of sixth, and radiated into the both carotids.

What is the interpretation?
The interpretation is aortic valve stenosis.

Examination of the precordium had shown that the patient had normal chest shape, no obvious scar, but there is wide area of epigastric pulsation. The apex beat was localised to the fifth intercostal space at the left midclavicular line, tapping in nature, and palpable P2, but no thrill or heaves. There was loud first heart sound, loud second heart sound, and systolic murmur as well as diastolic murmur. The systolic murmur is pansystolic, maximally localised at the left parasternal area, of blowing quality, no radiation, and third grade out of sixth while the diastolic murmur was mid-diastolic, maximally localised at the mitral area, rumbling quality, heard by bell of the stethoscope, and of second out of fourth grade.

What is the interpretation?
The interpretation is mitral stenosis complicated by pulmonary hypertension (tricuspid regurgitation)

Examination of the precordium had shown that the patient had normal chest shape, no obvious scar, but there is limited area of epigastric pulsation. The apex beat was localised to the fifth intercostal space at the left midclavicular line, normal apex beat, no palpable heart sounds, systolic thrill at the pulmonary area, with no lifts. There was normal first heart sound, fixed splitting of second heart sound, and systolic murmur. The murmur was ejection systolic murmur, maximally localised at the pulmonary area, harsh in quality, second grade, and radiated into the left shoulder.

What is the interpretation?
The Interpretation is ASD

Examination of the precordium had shown that the patient had pigeon chest shape, median sternotomy scar, and visible pulsation over both carotids and epigastric area. The apex beat was displaced to the sixth intercostal space at the left anterior axillary line, diffused and non-sustained with palpable A2, no thrill, and no lifts. There was soft first heart sound, loud metallic second heart sound, and diastolic murmur. The murmur was early diastolic, maximally localised at left parasternal area, of blowing quality, third grade out of sixth, and diffuse.

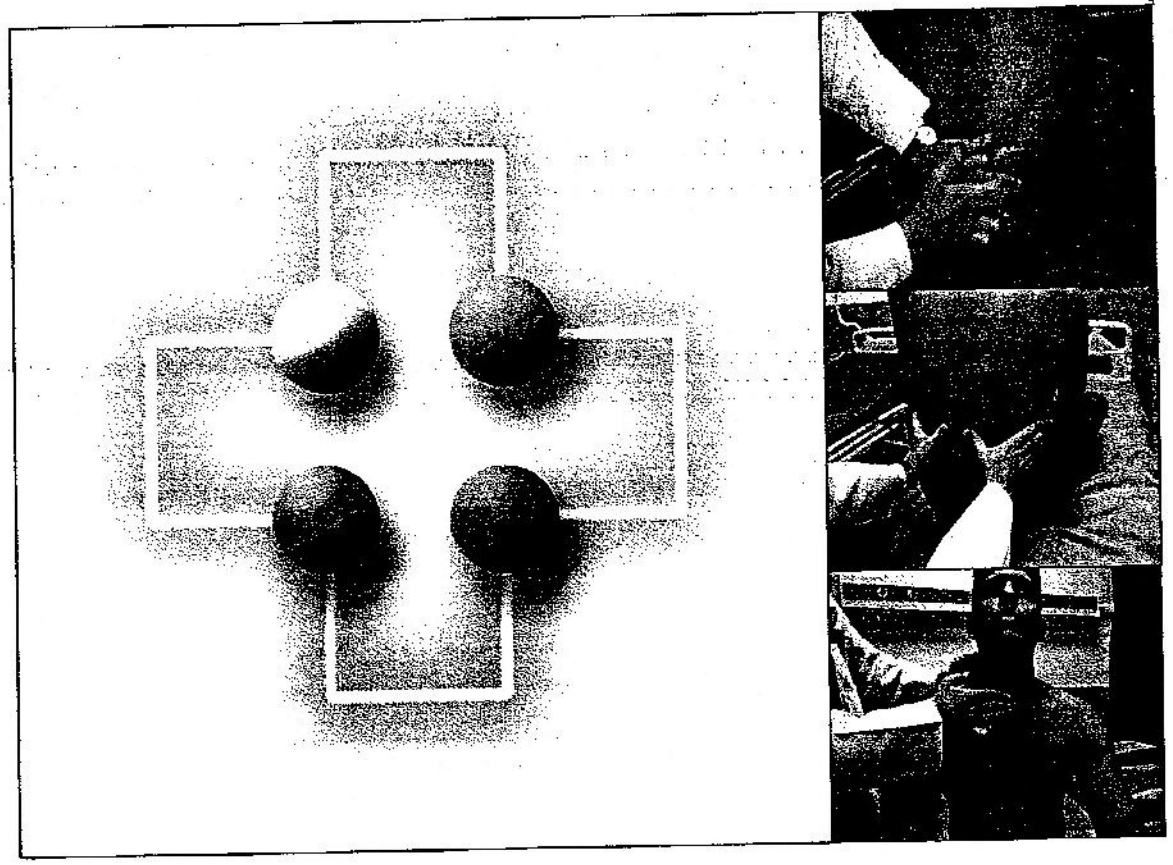
What is the interpretation?
The interpretation is Marfan's syndrome; pigeon chest, aortic valve prosthesis with regurgitation.

Further reading

- Hutchison's clinical methods
- Davidson's principles and practice of medicine
- Harrison's principles of internal medicine
- Cecil principles of internal medicine
- Macleod's clinical examination
- Oxford Handbook of Clinical Examination – Practical Skills
- First Aid for the Internal Medicine Boards
- Clinical Examination - A Systematic Guide to Physical Diagnosis
- Textbook of Clinical Neurology
- Queen Square Textbook of Neurology
- Dictionary of Neurological Signs
- Clinical Neurology (McGraw-Hill, 2009)
- 2005 Adams and Victor's Principles of Neurology
- Baily and Love's short practice of surgery
- Gynecology by ten teachers
- Obstetrics by ten teachers
- Hamilton Baily's physical signs in clinical surgery
- Browse; signs and symptoms of surgical diseases
- Nelson's text book of pediatrics
- Bate's guide to physical diagnosis.
- Textbook of physical diagnosis.
- Oxford handbook of clinical examination/ practical skills.
- Kochar's Clinical Medicine for Students.

5

RESPIRATORY SYSTEM



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ANATOMY OF THE RESPIRATORY SYSTEM

Thorax is that part of the body which contains the heart and the great vessels together with the lungs. It continues superiorly with the region of the neck by the superior aperture and separated inferiorly from the abdominal cavity by the diaphragm. The wall of the thorax is composed of the sternum which is situated anteriorly in the midline; the twelve thoracic vertebrae which are situated posteriorly in the midline; and the ribs which extend from the posterior aspect to the anterior aspect of the thoracic wall.

THE RIBS

The ribs are classified into true ribs and false ribs. The first seven ribs are the true ribs and attached directly to the sternum by their costal cartilages. The last five ribs are the false ribs and these ribs do not reach the sternum directly; the eighth, ninth and tenth ribs are attached to the costal cartilage of the seventh rib, while the eleventh and twelfth ribs are floated and do not reach to the sternum; for this reason they are called the floating ribs.

THE STERNUM

The sternum consists of 3 parts; the upper part is called the manubrium sternii; the middle part is the body of the sternum; and the lower part is the xiphoid process. The manubrium sternii and the body of the sternum attached to each other at an angle known as (angle of Louis) or (sternal angle) that receives the articulation of the second rib on either sides of the chest wall. The xiphoid process is cartilaginous process during most of the life, but in elderly, it becomes ossified.

THE LUNG AND THE PLEURA

The lungs are paired structure specialized for gaseous exchange. They are situated on either sides of the mediastinum and covered by pleural sacs. The pleura are formed from two layers:

1. A parietal layer lines the thoracic cavity
2. A visceral layer covers the lung tissue

The two layers are separated from each others by pleural cavity which is a potential space containing thin film of fluid to moisten the surface and to prevent friction. The two layers of the pleura are attached to each other at the margins of the hilum of the lung. In the hilum of the left lung, the bronchus is situated in the middle between pulmonary artery and pulmonary vein while in the hilum of the right lung, the pulmonary artery is situated in the middle between the bronchus and the pulmonary vein.

Each lung is expandable and spongy organ. The right lung is shorter and broader than the left lung. The right lung is divided into three lobes; the superior; the middle; and the inferior lobe by the oblique and horizontal fissures. The oblique fissure is situated at the level of the fifth rib, while the horizontal fissure is situated at the level of the fourth rib. The left lung is only divided into two lobes; the superior and the inferior lobe by the oblique fissure. Oblique fissure is also situated at the level of the fifth rib. Below this fissure and in the left lung only, the lower part of the lung forms a tongue-like process known as lingula. The left lung also characterized by the presence of cardiac notch which accommodates the left ventricle of the heart.

OPENINGS IN DIAPHRAGM

Through the diaphragm the following structures pass:

1. Inferior vena cava (8 letters) passes through the diaphragm at the level of T8
2. Oesophagus (10 letters) passes through diaphragm at the level of T10 together the right and left vagi
3. Aortic hiatus (12 letters) passes at the level of T12 together with the a Azygos vein and thoracic duct

RESPIRATORY TRACT

The surface area of both lungs is greater than 500m^2. The upper respiratory tract includes the nose, nasopharynx and larynx. It is lined by vascular mucous membranes with ciliated epithelium on their surfaces. Lower respiratory tract includes the trachea and bronchi. The trachea is a relatively thin walled semi rigid tube, about 4.5 inches long and 1 inch wide. The trachea bifurcates into two main (chief) bronchi at the level of sternal angle. There are 16-20 tracheal cartilages each is shaped C or Y; they nearly encircle the trachea, but open posteriorly. The trachea is divided into left and right main bronchi. The right main bronchus is shorter, wider and more vertical than the left. Thus inhaled foreign body is directed more commonly to the right main bronchus. Each bronchus enters the lung through the hilum and continues to divide into secondary bronchi and then tertiary bronchioles that give rise to the gas exchange unit of the lung which is the acinus. The acinus comprises branching respiratory bronchiole, alveolar ducts, alveolar sacs and clusters of alveoli. The alveoli are lined mostly with flattened epithelial cells (type I pneumocytes), but there are some, more cuboidal, type II pneumocytes. The latter produce surfactant, a mixture of phospholipids, which acts to reduce surface tension and counteract the tendency of alveoli to collapse. Type II pneumocytes also display a remarkable capacity to divide and reconstitute type I pneumocytes after lung injury. The total number of alveoli is 700,000,000.

ANATOMICAL LANDMARKS

The anatomical landmarks of the chest can be remembered as there are seven lines and seven points (shown below). To locate findings around the circumference of the chest, use a series of vertical lines, shown in FIGURE 5 – 1.

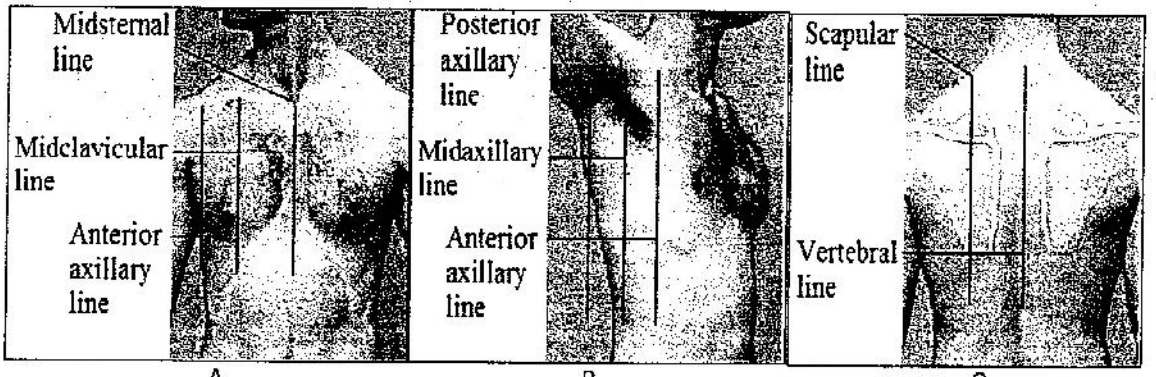


FIGURE 5 – 1. The vertical lines of the chest in an anterior view (A), right anterior oblique view (B), and posterior view (C).

You must be able to count the ribs and intercostal spaces. The sternal angle, also termed the angle of Louis, is the best guide: place your finger in the hollow curve of the suprasternal notch, and then move your finger down about 5 cm to the horizontal bony ridge joining the manubrium to the body of the sternum (FIGURE 5 – 2 A) then move your finger laterally and find the adjacent second rib and costal cartilage. From here, using two fingers, you can count (walk down the intercostal spaces), one space at a time, on an oblique line (FIGURE 5 – 2 B). Do not try to count intercostal spaces along the lower edge of the sternum; the ribs there are too close together. In a woman, to find the intercostal spaces either displace the breast laterally or palpate a little more medially than illustrated. Note that the costal cartilages of the first seven ribs articulate with the sternum; the cartilages of the eighth, ninth, and tenth ribs articulate with the costal cartilages just above them. The eleventh and twelfth ribs, the floating ribs, have no anterior attachments. The cartilaginous tip of the eleventh rib can usually be felt laterally, and the twelfth rib may be felt posteriorly. On palpation, costal cartilages and ribs feel identical.

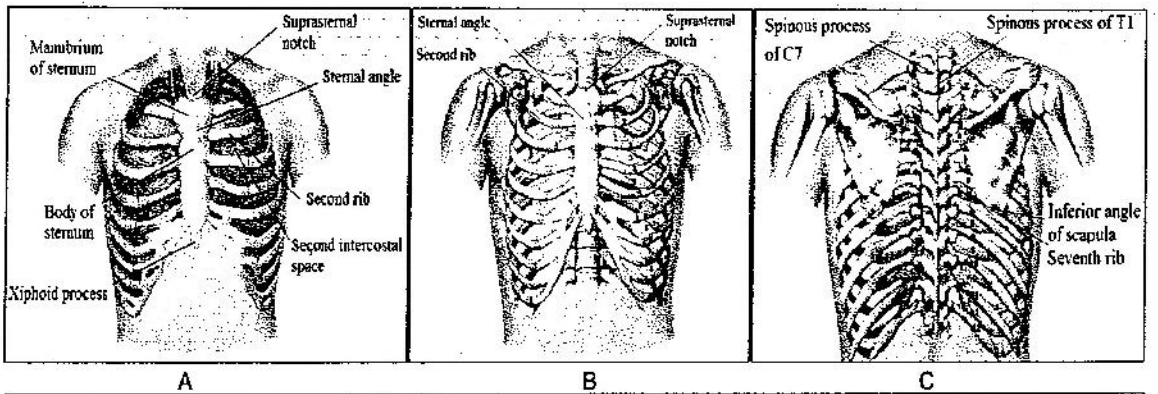


FIGURE 5 – 2. Anatomical landmarks of the chest including the location of angle of Louis (A) from which one can count the rib (B) and the twelfth rib posteriorly (C).

Posteriorly, the twelfth rib is another possible starting point for counting ribs and intercostal costal spaces: it helps locate findings on the lower posterior chest and provides an option when the anterior approach is unsatisfactory. With the fingers of one hand, press in and up against the lower border of the twelfth rib, then (walk up) the intercostal spaces or follow a more oblique line up and around to the front of the chest (FIGURE 5 – 2 C). The inferior tip of the scapula is another useful bony marker (FIGURE 5 – 2 C). It usually lies at the level of the seventh rib or intercostal space. The spinous processes of the vertebrae are also useful anatomic landmarks. When the neck is flexed forward, the most protruding process is usually the vertebra of C7. If two processes are equally prominent, they are C7 and T1. You can often palpate and count the processes below them, especially when the spine is flexed (FIGURE 5 – 2 C).

Anteriorly, the apex of each lung rises about 2 cm to 4 cm above the inner third of the clavicle (FIGURE 5 – 3). The lower border of the lung crosses the sixth rib at the midclavicular line and the eighth rib at the mid-axillary line. Posteriorly, the lower border of the lung lies at about the level of the T10 spinous process (FIGURE 5 – 3). The trachea bifurcates into its main stem bronchi at the levels of the sternal angle anteriorly and the T4 spinous process posteriorly. This is shown in FIGURE 5 – 3.

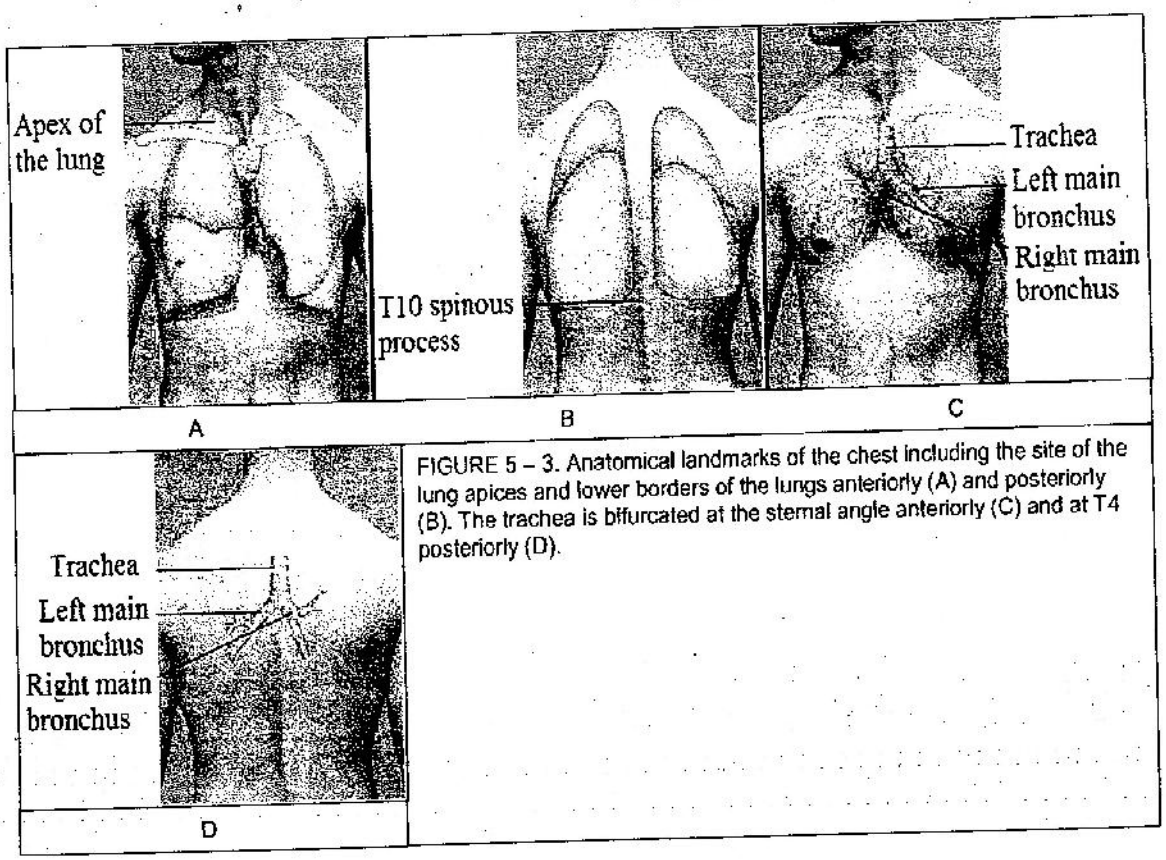


FIGURE 5 – 3. Anatomical landmarks of the chest including the site of the lung apices and lower borders of the lungs anteriorly (A) and posteriorly (B). The trachea is bifurcated at the sternal angle anteriorly (C) and at T4 posteriorly (D).

LOCATIONS ON THE CHEST

Be familiar with general anatomic terms used to locate chest findings, such as:

Supraclavicular	Above the clavicles	Interscapular	Between the scapulae
Infraclavicular	Below the clavicles	Infrascapular	Below the scapula

EXAMINATION OF THE RESPIRATORY SYSTEM

The clinical findings should be presented according to the lung lobes or lung zones as follows:

I According to the anatomical lung lobes (FIGURE 5 – 4) (more accurate method)

Each lung is divided roughly in half by an oblique (major) fissure. This fissure may be approximated by a string that runs from the T3 spinous process obliquely down and around the chest to the sixth rib at the midclavicular line

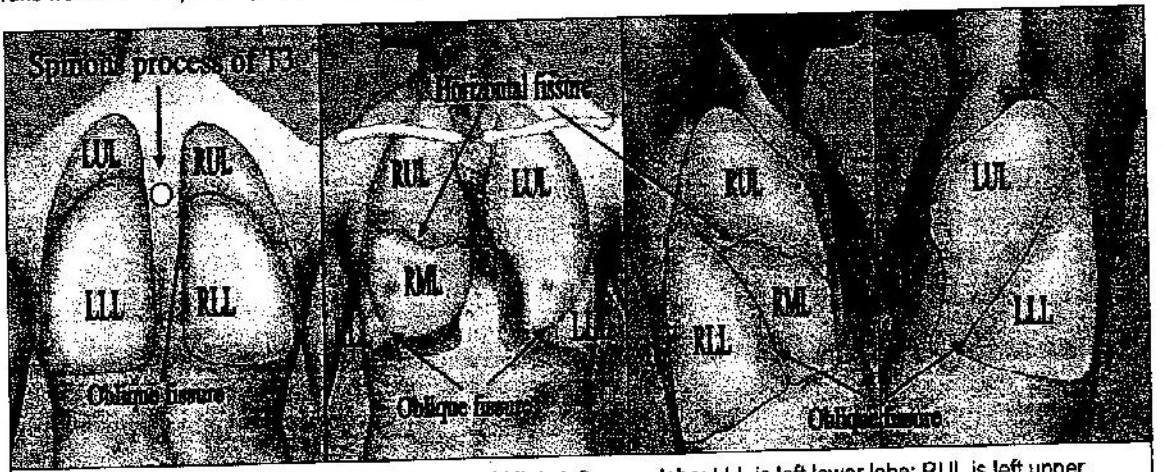


FIGURE 5 – 4. Lobes and fissures of the lungs. LUL is left upper lobe; LLL is left lower lobe; RUL is left upper lobe; RLL is left lower lobe; RML is right middle lobe.

The right lung is further divided by the horizontal (minor) fissure. Anteriorly, this fissure runs close to the fourth rib and meets the oblique fissure in the mid-axillary line near the fifth rib. The right lung is thus divided into upper, middle, and lower lobes. The left lung has only two lobes; upper and lower.

I According to the lung zones (FIGURE 5 – 5)

In this way (less accurate method); the lungs are divided into upper, middle and lower zones. The axillae are divided into upper axillary zones and lower axillary zones.

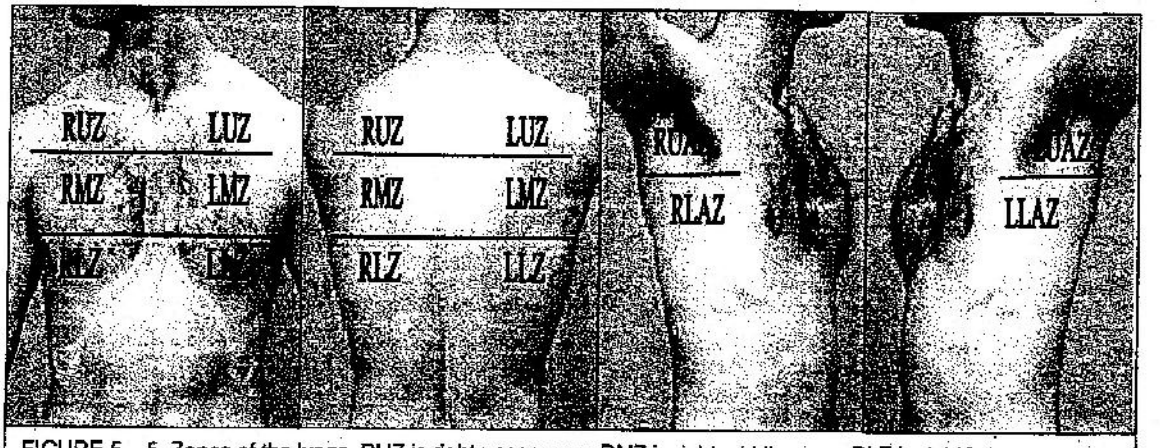


FIGURE 5 – 5. Zones of the lungs. RUZ is right upper zone; RMZ is right middle zone; RLZ is right lower zone; LUZ is left upper zone; LMZ is left middle zone: LLZ is left lower zone; RUAZ is right upper axillary zone; RLAZ is right lower axillary zone: LUAZ is left upper axillary zone; LLAZ is left lower axillary zone.

Upper zones lie above the sternal angle and the second rib anteriorly and above T4 posteriorly. The middle zones lie between the upper zones and above the fourth ribs anteriorly and T7 posteriorly. The lower zones lie below the middle zones and the sixth ribs anteriorly and T10 posteriorly.

The examination of the respiratory system includes general examination peculiar to the respiratory system, examination of anterior chest, and examination of posterior chest. The examination of the anterior or posterior chest should include inspection, palpation, percussion, and auscultation.

PHYSIOLOGY OF BREATHING

Most women make more use of the intercostal muscles than of the diaphragm, and therefore respiratory movements are predominantly thoracic. Men rely more on the diaphragm and their respiratory movements are predominantly abdominal (BOX 5 – 1). During inspiration, the diaphragmatic muscles contract, the central tendon moves down and the space between the diaphragm and the chest wall unfolds. The space thus created is filled by the lung. In thoracic respiration (in women), the chest cage is actively elevated by the external intercostal muscles and expanded by the action of the obliquely placed ribs. Because its structure is elastic, the chest cage returns to its resting position passively after expiration. During dyspnea other muscles will work. The chest cage is further elevated during inspiration by accessory muscles of inspiration. These are the scaleni, sternocleidomastoid, platysma and pectoral muscles. During expiration, the diaphragm relaxes and is pushed up by the intrabdominal pressure. The muscles of the abdominal wall may enhance this movement by pressing on the abdomen. The diaphragm is also depressed actively by the accessory muscles of expiration. These are the internal intercostal and the latissimus dorsi muscles.

BOX 5 – 1. Muscles of respiration	
Normal inspiration	Normal expiration
Diaphragm (male) External intercostal muscles (female)	Passive recoiling of thoracic cage
Dyspnoea	Dyspnoea
Scaleni Sternocleidomastoids Platysma Pectoral muscles	Internal intercostal muscles The latissimus dorsi Abdominal wall muscles causes increased abdominal pressure which pushes the diaphragm upward

EXAMINATION OF THE ANTERIOR CHEST

The examination of the anterior chest can be performed with the patient in semi-sitting and in sitting positions.

INSPECTION

Stand firstly at the end of the bed facing the patient to determine chest symmetry, shape of the chest, chest expansion, work of breathing (dyspnea), texture of the chest, and listen for any heard sound. After that stand beside the patient to count the respiratory rate, look at the chest texture, and listen further for any heard sound.

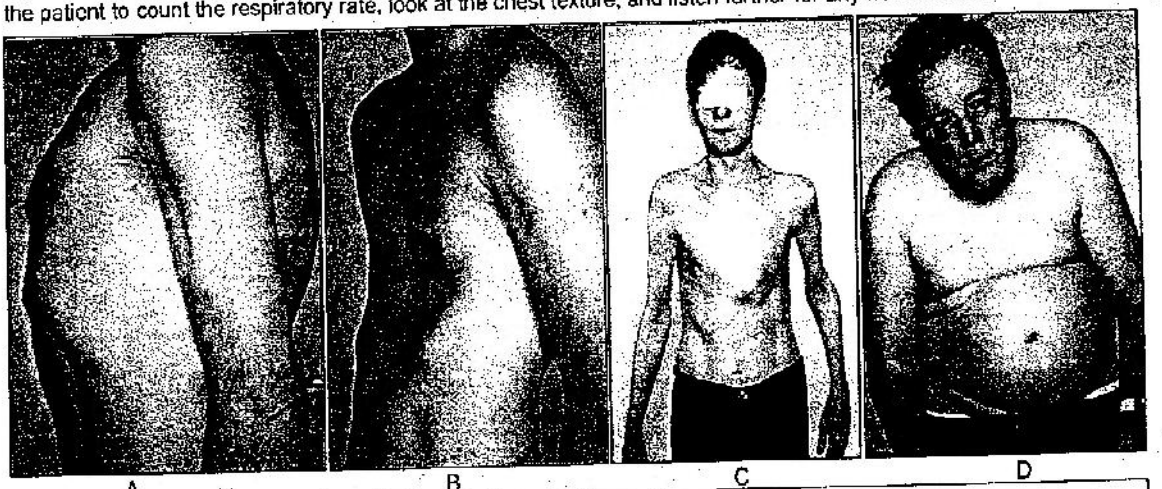


FIGURE 5 – 6. Abnormalities of the chest shape including barrel chest (A) pectus excavatum (B) pectus carinatum (C), and scoliosis (D) of the chest.

SYMMETRY AND SHAPE OF THE ANTERIOR CHEST

The anterior chest wall should be symmetrical, expanding well with inspiration, and recoiling with expiration. The normal shape of the chest is bilaterally symmetrical and elliptical in cross section with a transverse diameter that is more than anteroposterior diameter in a ratio 7:5 and there is no bending to one side. Abnormalities of the shapes of the chest had been described earlier in the cardiovascular system examination (FIGURE 5 – 6).

EXPANSION OF THE CHEST

Inspection of chest expansion is a traditional way to assess diminished or absence of chest inflation. Unilateral defect is easier to be notified than bilateral defect as the latter needs to be compared with normal chest expansion.

PATTERN OF BREATHING

Most women's respiratory movement is predominantly thoracic; men's respiratory movement is predominantly abdominal. Fast, deep breaths are seen in anxiety states. Deep, sighing breaths occurs in systemic acidosis and called acidotic breathing or Kussmaul's respiration (BOX 5 – 2).

BOX 5 – 2: Causes of Kussmaul breathing

KUSSMAUL

K: ketosis (diabetes, alcoholism, malnutrition)	A: aethylene glycol poisoning
U: uremia	U: uremia
SS: salicylate poisoning	L: lactic acidosis
M: methanol poisoning	

Cheyne-Stokes breathing describes irregular breathing with intermittent periods of increased rates and depths of breaths alternating with periods of apnea. This type of breathing is due to failure of the normal respiratory regulation in response to blood CO_2 levels and may occur in drug-induced respiratory depression, congestive heart failure, and brain damage (usually at the cerebral level). Prolonged expiratory phase of breathing is a marker of outflow limitation and suggests lung disease if coupled with pursed-lip breathing.

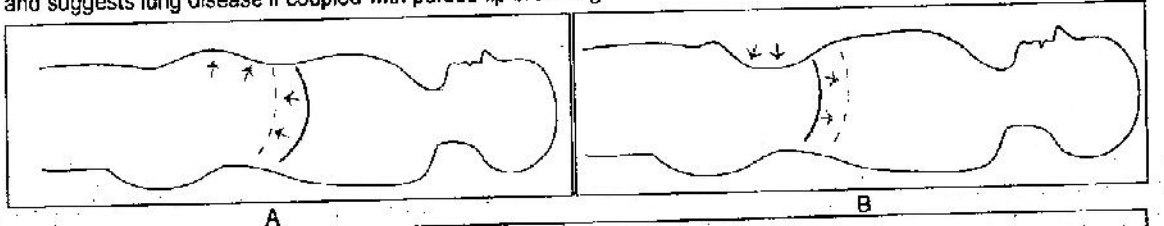


FIGURE 5 – 7. Normal movement of the diaphragm and abdominal contents with breathing (A) and abnormal, inward movement of the abdomen with each inspiratory effort (B). This is called abdominal paradox.

Look for paradoxical inward motion of the abdomen during inspiration (Hoover's sign) when the patient is supine (FIGURE 5 – 7). Abdominal paradox is an abnormal breathing pattern seen in patients who have diaphragm fatigue. It is recognized by observing the abdomen sink inward with each inspiratory effort. Fatigue of the diaphragm, which is the major muscle for breathing, occurs when it has been overworked and underfed. These circumstances are common in patients with acute exacerbations of COPD. The abdomen sinks inward with each breath because the negative intrathoracic pressure created by the accessory respiratory muscles causes the diaphragm and abdomen contents to be pulled upward into the chest during inspiration. This, in turn, causes the flaccid abdomen to sink inward during each respiratory inspiration.

DYSPNOEA

Dyspnoea or breathlessness is an undue awareness of breathing. It is normal with strenuous physical exercise. It can be defined clinically as shortness of breath with some or all of the following:-

I. Abnormal increased inspiratory movement evident by:

- Dilation of alae nasi.
- Indrawing of suprasternal fossa, supraclavicular fossae, and intercostals spaces due to the use of accessory muscles (scaleni, sternomastoids, platysma and pectoral muscles) of respiration to increase chest expansion during each inspiration. These muscles cause elevation of the shoulders with inspiration and aid respiration by increasing chest expansion. The use of accessory muscles is characteristic of patients with chronic obstructive pulmonary disease (COPD) and severe acute asthma.
- Abdominal paradox (Hoover's sign) that is indicative of diaphragmatic muscle fatigue.
- Tracheal descent during inspiration (tracheal tug).

II. Abnormal increased expiratory movement evident by:

- The patients assume upright or sitting position grasping a bed table or the back of the chair (Tripod position). This position stabilizes the shoulder girdles and helps the action of the accessory muscles.
- The patients exhale through their mouth with pursed lip and this is called (pursed lip breathing). Pursed-lip breathing provides a positive intra-airway pressure that is thought to decrease airway closure and air trapping and to aid gas exchange. Patients often discover this maneuver on their own or learn it through formal instruction by healthcare providers, although the exact benefit of this technique has not been established.

RESPIRATORY RATE

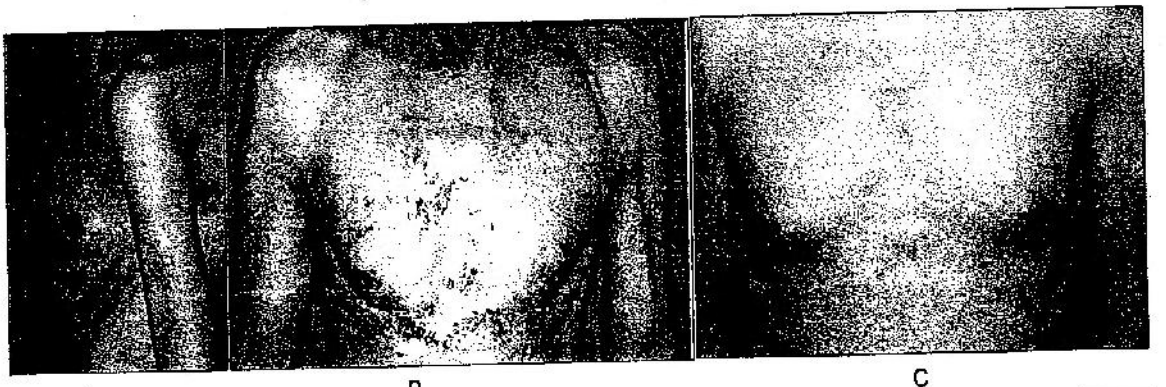
The normal rate of respiration in a relaxed adult is about 14 – 18 breaths (cycles) per minute. It is advisable to count the respiratory rate while your hand at the radial pulse so as to withdraw the attention of the patient. Respiratory rate more than 20 breaths (cycles) per minute is called tachypnea; less than 10 breaths (cycles) per minute is called bradypnea; Complete cessation of respiration is called apnea. The causes are shown in chapter three/vital signs examination.

HEARD SOUNDS

1	Wheeze	It is a musical sounds produced by the passage of air through narrowed bronchi. It is predominantly expiratory sounds. Wheeze is produced due to bronchial asthma, chronic obstructive pulmonary diseases, bronchiectasis, pneumonia, left ventricular failure, microscopic polyangiitis, eosinophilic lung disease, tumor causing partial obstruction and foreign body inhalation.
2	Strider	It is a harsh crowing sound produced by partial obstruction of large airways e.g. trachea, larynx. It is predominantly inspiratory sounds and occurs in laryngeal edema, laryngeal infection, and foreign body or tumor partially obstructs the trachea or larynx.
3	Hoarseness	It refers to an altered quality of the voice, often described as husky, rough, or harsh and may vary from slight harshness (dysphonia) to complete loss (aphonia) of voice. Hoarseness usually arises from disease of the larynx, but may also develop when extra-laryngeal lesions press on the laryngeal nerves e.g. a change in voice in a patient with lung cancer suggests invasion of the left recurrent laryngeal nerve at the hilum. In such cases the left vocal cord cannot adduct to the midline, resulting in failure to produce a normal explosive cough. The cough is then prolonged, low pitched and bovine.

TEXTURE OF THE CHEST (FIGURE 5 – 8)

Harrison's sulcus is a linear depression of the lower ribs just above the costal margins at the site of attachment of the diaphragm. It can result from severe asthma in childhood, or rickets. Subcutaneous emphysema is a crackling sensation felt on palpating the skin of the chest or neck. On inspection, there is often diffuse swelling of the chest wall and neck. It is caused by air tracking from the lungs and is usually due to a pneumothorax. Prominent veins may be seen in patients with superior vena caval obstruction. It is important to determine the direction of blood flow. Surgical stoma such as chest tube also be noted. Chest tube is used in pneumothorax, hemothorax, pyothorax, and hemopneumothorax. Scars or incisions of pleural biopsy or pleural aspiration are usually sited at the back of the patient at area of maximum dullness (aspiration of pleural effusion). Other abnormalities including tattooing, spider navi, gynecomastia, or primary or secondary skin lesions should also be noted.



A B C

FIGURE 5 – 8. Abnormalities of the chest texture including Harrison's sulcus (A) dilated superficial veins in SVC obstruction (B), and gynecomastia (C).

PALPATION

TENDERNESS

Ask the patient about any tenderness while you place your hands flat over chest. Causes of chest tenderness are:

1. Skin infection e.g. herpes zoster, cellulites
2. Injury to the chest involving skin, muscles, and bone fracture
3. Pulmonary causes such as malignancy involving the ribs and pleurisy
4. Cardiac problem such as pericarditis

LOCALIZATION OF THE APEX BEAT

In healthy adult person, the apex beat is located at the fifth intercostal space at, or slightly medial to mid-clavicular line. The detail of this examination is described in page 154 of cardiovascular system examination.

AXILLARY LYMPH NODES

The detail of this examination is described in page 95 of the general examination.

TRACHEA

It is usually conducted by gently introducing the tip of the index finger into the suprasternal notch exactly in the midline and press gently against the trachea (FIGURE 5 – 9). The middle finger may be used to localize the trachea with additional placement of the right ring finger on the attachment site of the sternomastoid muscle with the clavicle and index finger on the other side. If the trachea is deviated, the finger will slip partially or completely past it on the side opposite to the displacement. Causes of tracheal deviation are provided in BOX 5 – 3.

A B

FIGURE 5 – 9. Examination of the position of the trachea

BOX 5 – 3. Causes of tracheal deviation

Towards the side of the lung lesion	Away from the side of the lung lesion	Upper mediastinal mass
Upper lobe or lung collapse Upper lobe or lung fibrosis Pneumonectomy	Tension pneumothorax Massive pleural effusion	Retrosternal goiter Lymphoma or lung cancer

TRACHEAL TUG

Stand beside the patient and place your fingers between the suprasternal notch and cricoid cartilage which should be normally three to four finger breadths during inspiration (FIGURE 5 – 10). Reduction in this distance suggests lung hyperinflation. In severe lung hyperinflation, fingers resting on the trachea move inferiorly with each inspiration.

CHEST EXPANSION

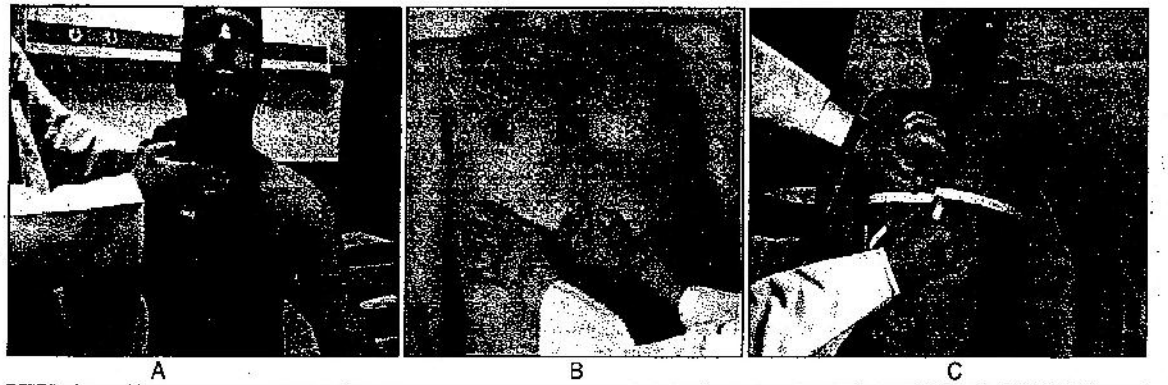
Normal chest expansion is 5 – 8 cm difference between full expiration and full inspiration. Chest expansion is performed in two ways (FIGURE 5 – 10):

1 With tape measure

Measure the chest of the patient using a tape measure encircling around the chest just below the nipples. A measured maximum chest expansion of less than 5 cm is probable abnormal and that of 2 cm or less is definitely abnormal

2 With both hands

Place the hands firmly on the chest wall with the fingers extending around the sides of the chest. The thumbs should almost meet in the middle line and should be lifted slightly off the chest so that they are free to move with respiration. As the patient takes a big breath in, the thumbs should move symmetrically apart at least 5 cm. Reduction of chest expansion to less than 5 cm is abnormal (BOX 5 – 4). Reduced expansion on one side indicates a lesion on that side

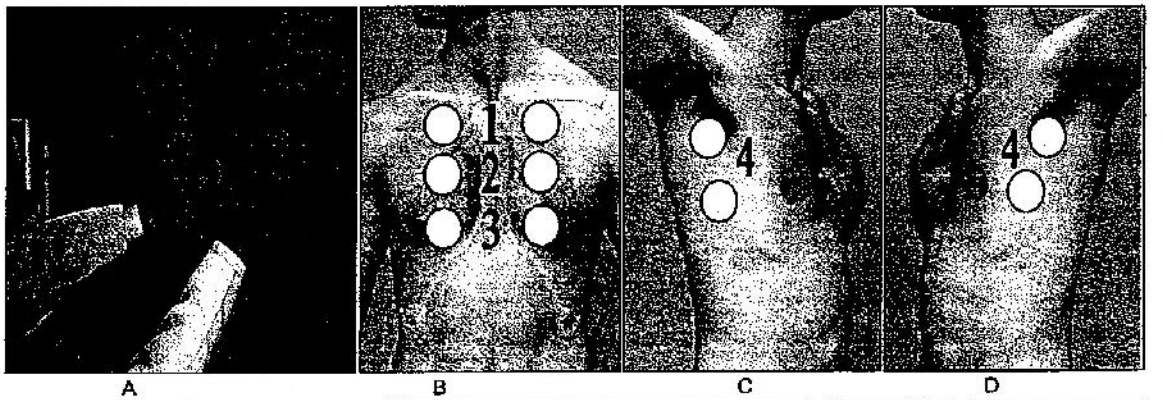


A B C

FIGURE 5 – 10. Techniques for tracheal tug (A), chest expansion with both hands (B), and chest expansion with tape measure (C).

BOX 5 – 4. Causes of diminished chest expansion

Unilateral diminished or absent expansion		Bilateral diminished or absent expansion
Consolidation	Pleural effusion	Bronchial asthma
Collapse	Pneumothorax	Emphysema
Fibrosis		Diffuse pulmonary fibrosis



A B C D

FIGURE 5 – 11. The techniques for vocal fremitus (A), and the areas you will place palm on (B, C, and D) which include (the numbers reflect the numbers in the FIGURES):

1. The upper lung zones (right and left)
2. The middle lung zones (right and left)
3. The lower lung zones (right and left)
4. The upper and lower axillary zones (right and left)

VOCAL (TECTILE) FREMITUS

Fremitus refers to the palpable vibrations transmitted through the bronchopulmonary tree to the chest wall when the patient speaks. To detect fremitus, place the bony part of the palm (at the base of the fingers) over the chest in the areas illustrated in FIGURE 5 – 11 while the patient repeat the number 44 (in Arabic). If fremitus is faint, ask the patient to speak more loudly or in a deeper voice. Practice is needed to appreciate the difference between normal and abnormal. Vocal fremitus may be absent in normal people (high-pitched voice or thick chest wall). It is only abnormal if different on one side from the other (BOX 5 – 5).

BOX 5 – 5. Causes of abnormal vocal fremitus

Increased vocal fremitus	Decreased vocal fremitus	
Consolidation	Pleural effusion	COPD
Fibrosis	Pneumothorax	

PERCUSSION

Percussion refers to tapping on a surface to determine the underlying structure. Tapping on the chest wall is transmitted to the underlying tissue, reflected back, and picked up by the candidate's tactile and auditory senses. The sound heard and the tactile sensation felt are dependent on the air-tissue ratio. The vibrations initiated by percussion of the chest enable the candidate to evaluate the lung tissue to a depth of only 5-6 cm, but percussion is valuable because many changes in the air-tissue ratio are readily apparent.

THE TECHNIQUE OF PERCUSSION (FIGURE 4 – 12)

1 STEP I

Place the left hand on the chest wall, palm downward, fingers slightly separated with the second phalanx of the middle finger over the areas to be percussed usually an intercostal spaces. The exception for this rule is that percussion over clavicles needs only right hand percussion without a pleximeter

2 STEP II

Firmly press the left middle finger against the chest wall and strike the centre of the second phalanx with the tip of the right middle finger held at a right angle to produce a hammer like effect with the entire movement coming form wrist joint and never forget to compare between right and left sides.

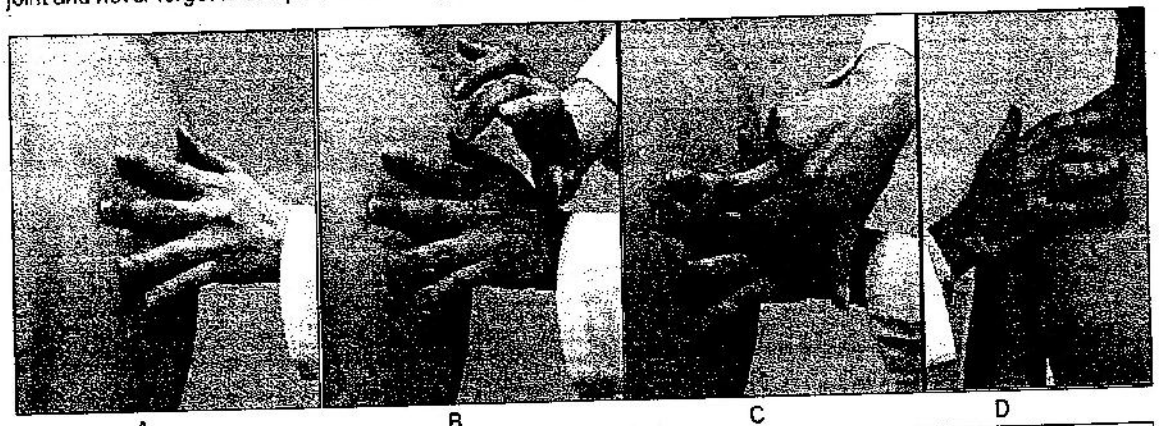


FIGURE 5 – 12. The techniques for percussion (A, B, and C) over lung tissue and over the clavicles (D). In the latter technique there is no need for pleximeter.

3 STEP III

Remove the percussing finger quickly so the note generated is not dampened

4 STEP IV

Percuss over the anterior chest and both axillae (FIGURE 5 – 13)

Percuss the lung apices by placing the palmar surface of your left middle finger across the anterior border of the trapezius muscle, overlapping the supraclavicular fossa and percussing downwards

Percuss the clavicle directly over the medial third, as percussing laterally is dull over the shoulder muscles.

5 STEP V

Compare each left tap with the right tap

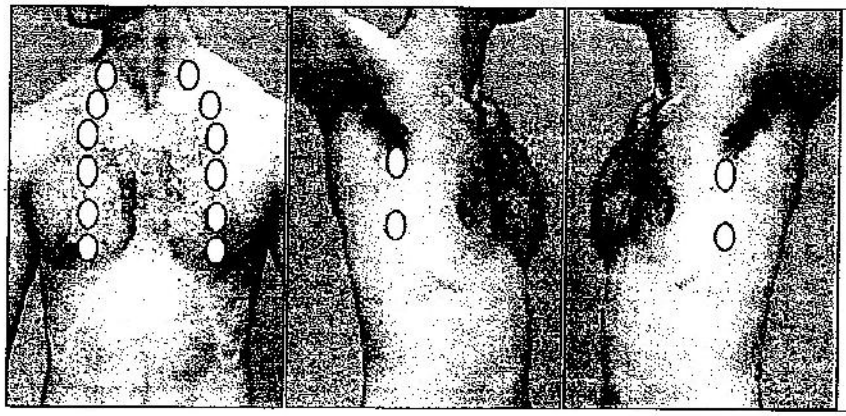


FIGURE 5 – 13. The sites over both lungs and axillae that need to be percussed are the same as that needed for auscultation with the exception of clavicles.

Percussion over a solid organ, such as the liver, produces a dull percussion note without resonance. Percussion over a structure containing airs within a tissue, such as the lung, produces a resonant percussion note. Percussion over a hollow air-containing structure, such as the stomach, produces a tympanic percussion note. Normally, in the chest, dullness is heard and felt over the heart and over the liver while the remaining areas should produce resonance percussion note. As the lungs fill with fluid and become denser, as in pneumonia, resonance is replaced by dullness. The term Hyper-resonant has been applied to the percussion note obtained from a lung with decreased density, such as that found in pneumothorax. Features of percussion notes are provided in BOX 5 – 6.

CLINICAL CLUE

Try percussion initially on yourself. Percuss over your right lung (resonant), stomach (tympanic), and liver (dull). In adults the upper level of liver dullness is the fifth rib in the midclavicular line. Resonance below this is a sign of hyperinflation (COPD or severe asthma).

BOX 5 – 6. Features of percussion notes

Resonant	Dull	Stony dull	Hyper-resonant
Normal lung	Consolidation Collapse Fibrosis	Pleural effusion Hemothorax Large hydatid cyst	Pneumothorax COPD

AUSCULTATION

Perform auscultation with the patient relaxed and breathing deeply and fairly rapidly through the mouth. Avoid prolonged deep breathing and auscultation in area less than 2 – 3 cm from midline. The examination should proceed from side to side and from top to bottom, one point being compared with the other. The auscultation points are illustrated in FIGURE 5 – 14.

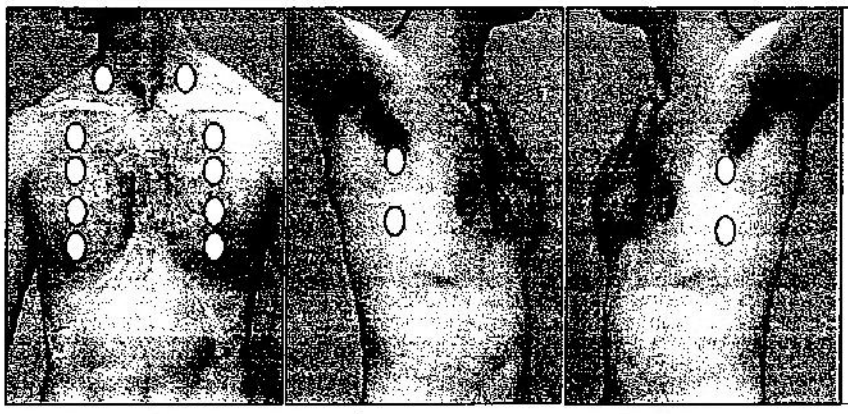


FIGURE 5 – 14. The sites over both lungs and axillae that need to be auscultated are the same as that needed for percussion with the exception of clavicles.

Because most breath sounds are high pitched, the diaphragm is used to evaluate lung sounds. The auscultation should be performed anteriorly from above the clavicle down to the sixth rib inferiorly and laterally from the axilla to the eighth rib (Why? These are the boundaries of the lungs). The examination sequence of auscultation is as follows:

1. Determine air entry to the lungs — Good air entry or diminished air entry or absent air entry
2. Determine the breath sound — Vesicular breathing or bronchial breathing or vesicular breathing with prolonged expiratory phase
3. Determine any added sounds. These are
   - Crackles
     - Fine or coarse
     - Inspiratory (early, middle, or late) or biphasic
     - Change with cough
   - Wheezes
     - Localised, diffuse, or scattered
     - Inspiratory, expiratory, or biphasic
     - Monophonic or polyphonic (for postgraduate students)
     - Low pitch or high pitch (for postgraduate students)
     - Change with cough
   - Pleural rub
   - Pneumothorax click
4. Perform vocal resonance

AIR ENTRY TO THE LUNGS

The candidates should ascertain the presence of breath sound. Diminished or absent air entry usually caused by pleural effusion or thickening, collapse, pneumothorax, or emphysema

BREATH SOUNDS

The breath sounds are heard over most of the lung fields. They consist of an inspiratory phase followed by an expiratory phase. Breath sound may take one of the following forms (FIGURE 5 – 15):

1. Vesicular breathing — This is normally heard over most of the healthy lung tissue and consists of full inspiratory phase and third expiratory phase and there is no gap between expiration and inspiration.
2. Bronchial breathing — Bronchial breath sounds are more easily remembered than described. They are audible in normal people, posteriorly over the right upper chest where the trachea is contiguous with the right upper bronchus. Other than this site this type of breath sound is considered abnormal (BOX 5 – 7). It consists of full inspiratory phase and full expiratory phase and there is a gap between them. Occasionally breath sounds over a large cavity have an exaggerated bronchial quality. This is called amphoric bronchial sound and has been likened to that heard when air passes over the top of a hollow jar (Greek amphoreus).
3. Vesicular breathing with prolonged expiratory phase — In which there is full inspiratory phase, no gap but prolonged expiratory phase. This type of breathing suggests asthma and COPD.

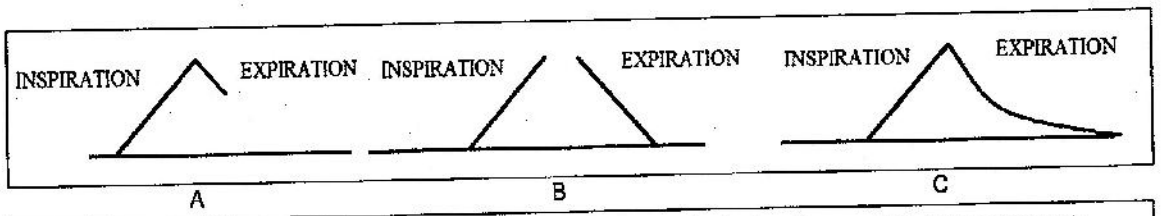


FIGURE 5 – 15. The types of the breath sounds including normal vesicular breath sound (A), bronchial breath sound (B), and vesicular breathing with prolonged expiratory phase (C).

BOX 5 – 7. Causes of bronchial breathing

Consolidation	Fibrosis
Collapse	Over pleural effusion

When bronchial breath sounds are heard, the candidate my be asked to perform two techniques called egophony and whispered pectoriloquy.

1. Egophony is said to be present when the spoken word heard through the lungs is increased in intensity and takes on a nasal or bleating quality. The patient is asked to say (eeee) as in 'bee' whiles the candidate listens to an area in which consolidation is suspected. If egophony is present, the (eeee) will be heard as (aaaa) as in 'bay'. This (e to a) change is seen in consolidation of lung tissue. The area of compressed lung above a pleural effusion often produces egophony.
2. Whispered pectoriloquy is the term for the intensification of the whispered word heard in the presence of consolidation of the lung. The patient is instructed to whisper (one-two-three) while the candidate listens to the area suspected of having consolidation. Normally, little or nothing may be heard when one listens to a normal chest. However, if consolidation is present, the transmission of the spoken words will be increased, and the words will be clearly heard.

ADDED (ADVENTITIOUS) SOUNDS

Adventitious sounds include the following:

Crackles (also called rales or crepitations) are short, interrupted, nonmusical sounds heard mostly during inspiration and are caused by the opening of collapsed distal airways and alveoli. A sudden equalization of pressure seems to result in a crackle. Coarser crackles are related to larger airways. Fine crackles are likened to the sound made by rubbing hair next to the ear or the sound made when Velcro is opened.

BOX 5 – 8. Causes of crackles according to the inspiratory phase

Phase of inspiration	Cause
Early crackles	Small airways disease (bronchiolitis) and early stages of pneumonia
Middle crackles	Pulmonary edema
Late crackles	Pulmonary fibrosis (fine) Bronchial secretions in COPD, resolving pneumonia, lung abscess, tubercular lung cavities (coarse)
Biphasic crackles	Bronchiectasis (coarse)

Crackles may be described as fine or coarse depending on their quality (BOX 5 – 8). Crepitations may be heard in healthy individual particularly at the base of the lung, but when such an individual asked to cough, crepitations will disappear.

Wheezes are continuous musical sounds heard mostly during expiration. Wheezes produced by airflow through narrowed bronchi by swelling, secretions, spasm, tumor, or foreign body. Wheezes should be described as expiratory, inspiratory, or biphasic. This is because the airways normally dilate during inspiration and are narrower during expiration. An inspiratory wheeze implies severe airway narrowing. In general, expiratory wheeze is usually due to bronchial asthma, or COPD. Inspiratory wheeze is usually due to infection, tumour or foreign body. Biphasic wheeze is usually due to infection and COPD. Wheezes should also be described as diffused (over all lung zones), localised, or scattered. Monophonic wheezes produce a single musical note that does not clear on coughing. A monophonic or localised wheeze which fails to clear with coughing suggests the presence of a fixed bronchial obstruction. High-pitched wheeze arises from smaller airways and has a whistling quality (often due to asthma) while low-pitched wheeze originates from larger bronchi (often due to COPD).

Pleural rub is a grating sound produced by motion of the pleura, which is impeded by frictional resistance. It is best heard at the end of inspiration and at the beginning of expiration and did not change with cough to be distinguished from crepitations. The sound of a pleural rub is like the sound of creaking leather. Pleural rubs are heard when pleural surfaces are roughened or thickened by inflammatory or neoplastic cells or by fibrin deposits. To differentiate pleural rub from pericardial rub, ask the patient to stop breathing; if the rub persists it is pericardial rub.

Pneumothorax click is a rhythmical sound synchronous with cardiac systole, produced when there is air between the two layers of pleura overlying the heart.

VOCAL RESONANCE

The same instructions applied for vocal fremitus are used here with the stethoscope.

FOR POSTGRADUATE STUDENTS

Check for forced expiratory time (FET) if your diagnosis is chronic obstructive airways disease (COAD) by asking the patient to exhale forcefully after full inspiration while you are listening over the trachea. The normal forced expiratory time is 3 seconds or less: if the patient takes more than 6 seconds, airway disease is likely.

Squeak is a musical sound heard in inspiration or in both inspiration and expiration. It is caused by diffuse pulmonary fibrosis especially if due to alveolar allergy e.g. hypersensitivity pneumonitis.

EXAMINATION OF THE CHEST POSTERIORLY

It is helpful to examine the posterior thorax and lungs while the patient is sitting. During percussion and auscultation, the patient's arms should be folded across the chest with hands resting, if possible, on the opposite shoulders. This position moves the scapulae partly out of the way and increases your access to the lung fields. The examination of the posterior chest, like anterior chest, includes inspection, palpation, percussion, and auscultation.

INSPECTION

Stand behind the patient facing his/her back and determine chest symmetry, shape of the chest, chest expansion, work of breathing (dyspnea), texture of the chest, and listen for any heard sound.

SYMMETRY AND SHAPE OF POSTERIOR CHEST
The posterior chest wall should be symmetrical, expanding well with inspiration, and recoiling with expiration. Determine whether the patient had normal chest shape or scoliosis, or kyphoscoliosis (FIGURE 5 – 16).

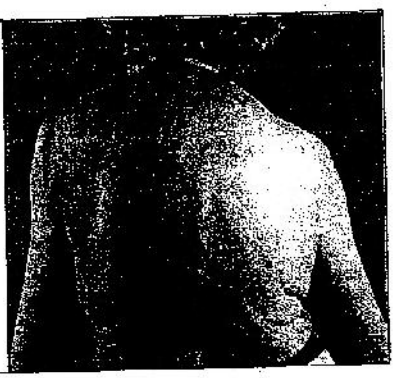


FIGURE 5 – 16. Kyphoscoliosis of the chest with lateral and forward bending.

EXPANSION OF THE CHEST
Expansion of the back should be symmetrical. Unilateral chest expansion defect is easier to be notified than bilateral defect as the latter needs to be compared with normal chest expansion.

PATTERN OF BREATHING
Prolonged expiratory phase of breathing is a marker of outflow limitation and suggests lung disease.

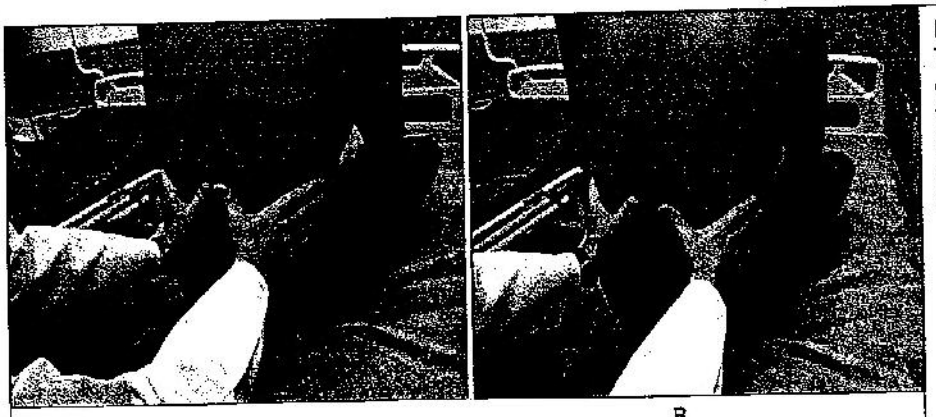
DYSPNOEA
Look for retractions of intercostals spaces and patient's position which may suggest dyspnoea.

HEARD SOUNDS
Listen for any sounds e.g. wheezes, stridor, or hoarseness.

TEXTURE OF THE CHEST
Look especially for incisions and scars of pleural biopsy or pleural aspiration which are usually sited at the back of the patient. Other abnormalities should also be noted.

PALPATION

TENDERNESS
Ask the patient about any tenderness while you place your hands flat over chest.



A B

FIGURE 5 – 17. The techniques for chest expansion using both hands during resting state (A) and during expiration (B).

CHEST EXPANSION

Normal chest expansion is 5 – 8 cm difference between full expiration and full inspiration. The same maneuvers applied to the anterior chest are also applicable to the posterior chest (FIGURE 5 – 17).

VOCAL (TECTILE) FREMITUS

The technique used for the anterior chest wall is also applicable for the posterior chest. The difference lays in the sites used to illicit the vocal fremitus (FIGURE 5 – 18).

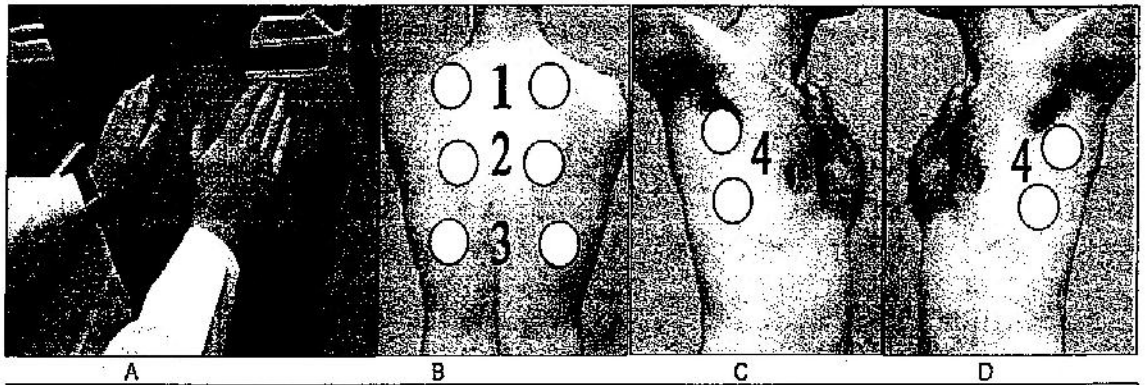


FIGURE 5 – 18. The techniques for vocal fremitus (A) and the areas you will place palm on (B, C, and D) which include (the numbers reflect the numbers in the FIGURES):

1. The upper lung zones (right and left)
2. The middle lung zones (right and left)
3. The lower lung zones (right and left)
4. The upper and lower axillary zones (right and left)

PERCUSSION

Percussion is performed in similar way to that described for anterior chest keeping in mind not to percuss near the midline (away 2 – 3 cm), as this produces a dull note from the solid structures of the thoracic spine and paravertebral musculature and not to percuss below T10 (Why? This is the lower border of the lung from the posterior aspect). The sites of percussion are shown in FIGURE 5 – 19.

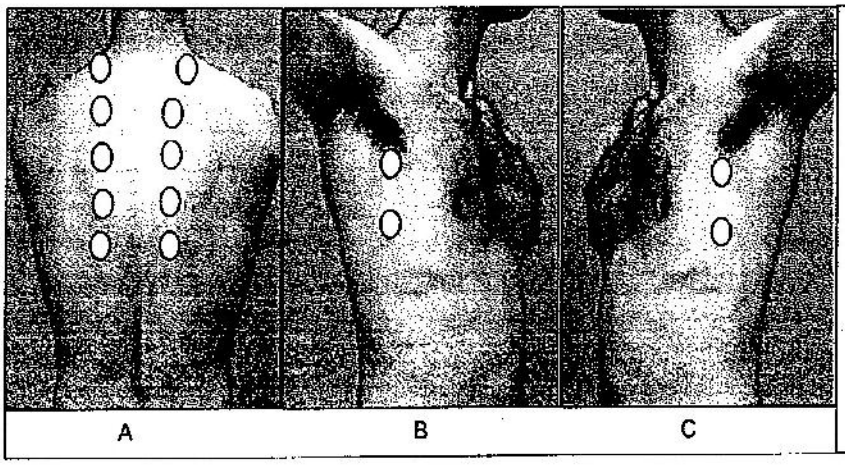


FIGURE 5 – 19. The sites over both lungs and axillae that need to be percussed are the same as that needed for auscultation.

DIAPHRAGMATIC MOVEMENT (EXCURSION)

Percussion is also used to detect diaphragmatic movement from the right side. The patient is asked to exhale as much as possible. Percussion is started from the upper part of the right side till the point where percussion note became dull which represent the lowest part of the diaphragm. The patient then is instructed to take deep breath and hold it. The same level of dullness will change now to resonant as the lung became full and the liver moves down. Below this level is dullness from the liver. The difference between the inspiration and expiration levels represents diaphragmatic motion, which is normally 4 – 5 cm. In patients with emphysema, the diaphragmatic motion is reduced. In patients with phrenic nerve palsy, diaphragmatic motion is absent. This test is illustrated in FIGURE 5 – 20.

AUSCULTATION

The techniques, the instructions, and the findings are the same as these mentioned for anterior chest examination. The sites used to listen for are the same points used for percussion.

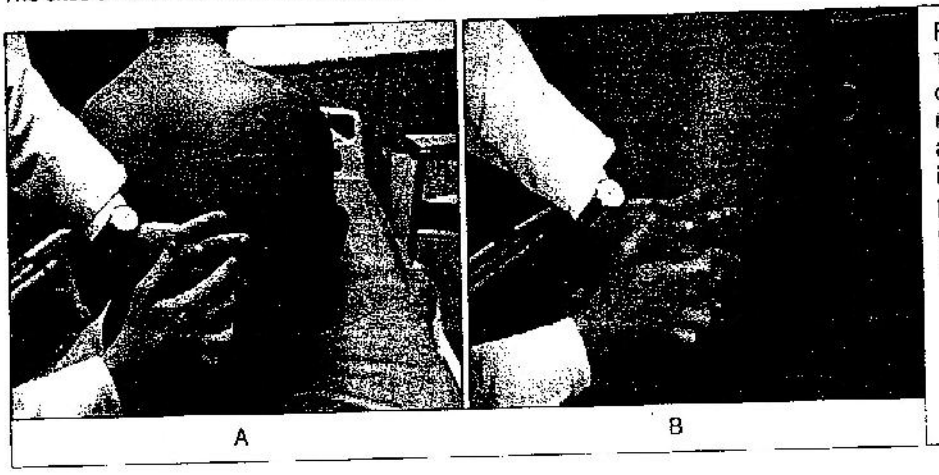


FIGURE 5 – 20. The technique of diaphragmatic motion assessment includes percussion during expiration (A) and inspiration (B).

EXAMPLES

Examination of the anterior chest of this patient had shown normal symmetrical elliptical chest shape with good expansion; no abnormal heard sounds, no dyspnoea, and the respiratory rate is 12 cycles per minute. The apex beat was localised to the left fifth intercostal space slightly medial to the midclavicular line; no palpable axillary lymph nodes; the trachea is centrally located with no tracheal tug; symmetrical vocal fremitus; chest expansion was 8 cm difference between inspiration and expiration. The percussion notes were resonant over anterior chest and both axillae apart from cardiac and liver dullness. There was good air entry with vesicular breathing heard over all the lung tissues, no added sounds, and normal symmetrical vocal resonance.

What is the interpretation?
The interpretation is normal chest examination.

Examination of the posterior chest of this patient had shown normal symmetrical elliptical chest shape with good expansion; no abnormal heard sounds, no intercostal recession, and the respiratory rate is 12 cycles per minute. There was symmetrical vocal fremitus with chest expansion of 8 cm difference between inspiration and expiration. The percussion notes were resonant over posterior chest and both axillae. There was good air entry with vesicular breathing heard over all the lung tissues, no added sounds, and normal symmetrical vocal resonance.

What is the interpretation?
The interpretation is normal chest examination.

Examination of the back of this patient had shown asymmetrical chest with reduction in the expansion of the right side; visible intercostal recession on the right side with small peace of surgical dressing applied at the lower zone of the chest posteriorly. There was reduced chest expansion on the right side as well as reduction of vocal fremitus; the percussion notes were resonant over the left side but stony dullness over the right side. Poor air entry, absent breathe sound and vocal resonance over the right side of the chest while the left side showed good air entry, normal vesicular breathing, and no added sounds.

What is the interpretation?
The interpretation is probably right sided pleural effusion.

Examination of the anterior chest of this patient had shown elliptical chest shape with reduction in the expansion of the right side; no heard sounds but there were dyspnoea and tachypnea. The apex beat was localised to the left fifth intercostal space slightly medial to the midclavicular line; no palpable axillary lymph nodes; the trachea is centrally located with no tracheal tug; vocal fremitus was asymmetrical and increased in the right middle and lower zone; chest expansion was reduced on the right side. The percussion notes were resonant over the left side of the chest apart from cardiac dullness but the notes were dull in the right middle and lower zones and right axilla. There was good air entry with vesicular breathing heard over the left side but bronchial breathing over the right middle and lower zones with egophony and pectoriloquy; late inspiratory crackles are also heard over the right side of the chest. The vocal resonance was asymmetrical and increased over the right side of the chest.

What is the interpretation?
The interpretation is probably consolidation of the right middle and lower zones.

Examination of the posterior chest of this patient had shown asymmetrical chest expansion with reduction on the left side, intercostal recession with each breath and respiratory rate of 20 cycles per minute and no abnormal heard sounds. There was asymmetrical vocal fremitus which increased over the left side; chest expansion was asymmetrical with reduction over the left side. The percussion notes were resonant over right posterior chest and axilla but dull notes over the left lower and middle zones. There was poor air entry over the left lower and middle zones; vesicular breathing heard over the right side of the back, no added sounds, and asymmetrical vocal resonance.

What is the interpretation?
The interpretation is probably left lobar collapse.

Examination of the anterior chest of this patient had shown normal barrel-shaped chest with increased anteroposterior diameter, symmetrical reduction in expansion; no abnormal heard sounds, dyspnoea and tachypnea with pursed lip breathing and use of accessory muscles of respiration and drawing in of the lower intercostal muscles with inspiration. The apex beat was difficult to be localised; no palpable axillary lymph nodes; the trachea is centrally located with tracheal tug; symmetrical reduction in vocal fremitus and chest expansion which was limited to two cm difference between inspiration and expiration. The percussion notes were hyper-resonant over anterior chest and both axillae with reduction in both cardiac and liver dullness. There was poor air entry bilaterally; vesicular breathing with prolonged expiratory phase; coarse late inspiratory crackles over all the lung tissues, and symmetrically reduced vocal resonance.

What is the interpretation?
The interpretation is probably COPD (emphysema).

Examination of the posterior chest of this patient had shown symmetrical reduction in chest expansion, increased in the anteroposterior diameter, wheezes, dyspnea and tachypnea, with obvious prolongation of the expiration. There was symmetrically reduced vocal fremitus and chest expansion which showed 2 cm difference between inspiration and expiration. The percussion notes were hyper-resonant over posterior chest and both axillae. There was poor air entry bilaterally, vesicular breathing with prolonged expiratory phase heard over all the lung tissues, inspiratory and expiratory diffuse wheezes. The vocal resonance was symmetrically reduced.

What is the interpretation?
The interpretation is probably bronchial asthma.

Examination of the anterior chest of this patient had shown reduced chest expansion on the right side, no abnormal heard sounds, dyspnoea and tachypnea. The apex beat was localised to the left fifth intercostal space displaced lateral to the midclavicular line; no palpable axillary lymph nodes; the trachea was displaced to the left side with no tracheal tug; vocal fremitus was reduced over the right side; obvious reduction in chest expansion on the right side. The percussion notes were resonant over the left side of the chest but hyper-resonant on the right side. There was good air entry on the right side; the type of breath sound was vesicular with no added sound. The right side showed absent air entry. The vocal resonance was markedly reduced over the right side.

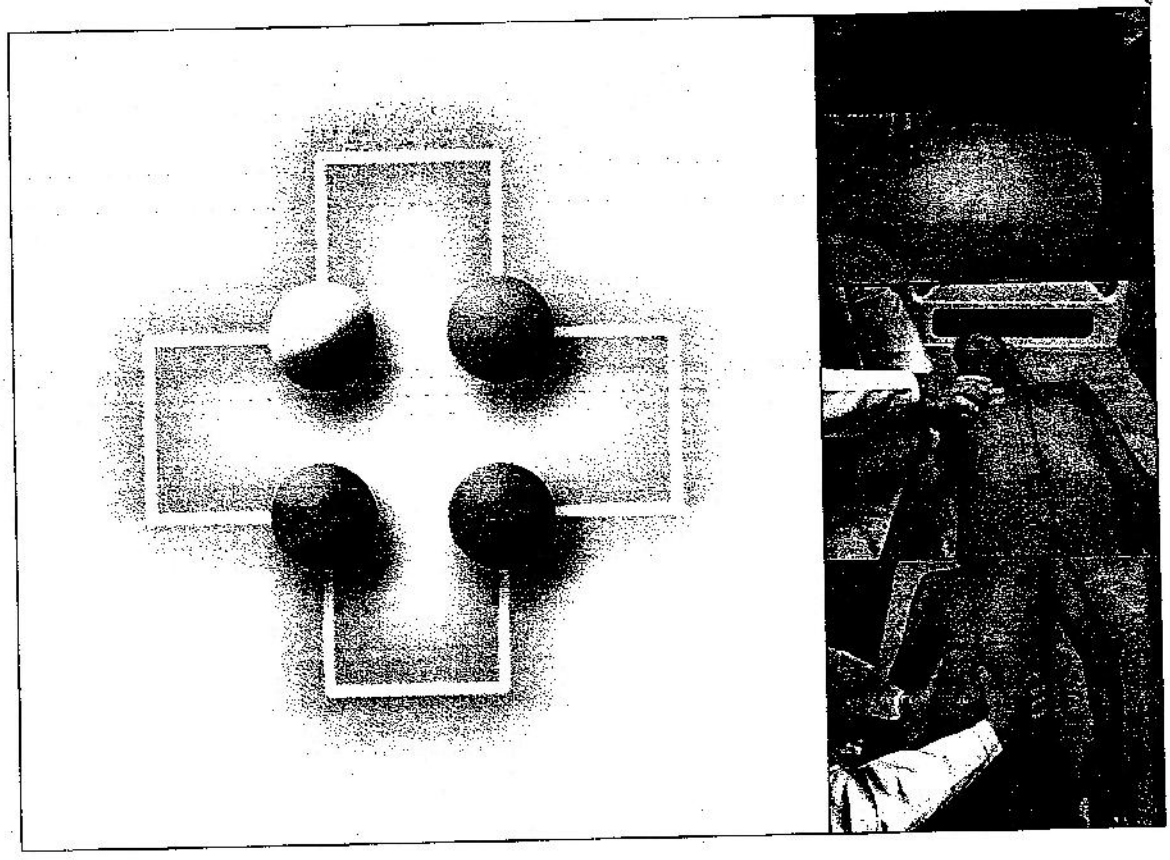
What Is the interpretation?
The interpretation is right pneumothorax.

Further reading

- Hutchison's clinical methods
- Davidson's principles and practice of medicine
- Harrison's principles of internal medicine
- Cecil principles of internal medicine
- Macleod's clinical examination
- Oxford Handbook of Clinical Examination – Practical Skills
- First Aid for the Internal Medicine Boards
- Clinical Examination - A Systematic Guide to Physical Diagnosis
- Textbook of Clinical Neurology
- Queen Square Textbook of Neurology
- Dictionary of Neurological Signs
- Clinical Neurology (McGraw-Hill, 2009)
- 2005 Adams and Victor's Principles of Neurology
- Baily and Love's short practice of surgery
- Gynecology by ten teachers
- Obstetrics by ten teachers
- Hamilton Baily's physical signs in clinical surgery
- Browse; signs and symptoms of surgical diseases
- Nelson's text book of pediatrics
- Bate's guide to physical diagnosis.
- Textbook of physical diagnosis.
- Oxford handbook of clinical examination/ practical skills.
- Kochar's Clinical Medicine for Students.

6

ABDOMEN EXAMINATION



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ANATOMY OF THE GASTROINTESTINAL TRACT

The digestive system comprises the mouth, pharynx and alimentary canal. In addition, it includes the salivary glands, pancreas and liver which open to these parts. The alimentary canal begins with the esophagus and ends with the anus.

THE ESOPHAGUS

This tube is approximately 25 cm long occupying the posterior mediastinum and extending from the cricopharyngeal sphincter to the cardia of the stomach. There is an upper and a lower esophageal sphincter. The upper sphincter is powerful skeletal muscle, while the lower sphincter is composed of smooth muscles. The main function of the esophagus is to transfer food from the mouth to the stomach.

THE STOMACH

The stomach is a tube that became dilated and distorted into potentially capacious sac. The main function of the stomach is to retain and grind food stuffs. The stomach is divided into four regions; the cardia, the fundus, the body, and the antrum.

THE SMALL INTESTINE

The small bowel extends from the ligament of Treitz to the ileocaecal valve. The small intestine is divided, without sharp limits, into three portions; the duodenum, the jejunum and the ileum. The main functions of the small intestine are digestion, absorption of the products of digestions, and protection against ingested toxins.

THE LARGE INTESTINE

The large intestine is divided into; the cecum, appendix, colon, rectum and anal canal. The main function of the large intestine is to absorb water and electrolytes.

ANATOMY OF THE UROLOGICAL SYSTEM

The urinary system consists of two kidneys, single, midline urinary bladder, two ureters (which carry urine from the kidneys to the urinary bladder), and single urethra (which carries urine from the bladder to the outside of the body).

KIDNEYS

The kidneys are bean-shaped organs lying on the posterior abdominal wall behind the peritoneum. The superior pole of the right kidney is slightly lower than the left because of the presence of the liver superior to it. Each kidney measures about 12 cm long, 6 cm wide and 3 cm thick. On the medial side of each kidney there is a relatively small area called the hilum where the renal artery and the nerves enter and the renal vein and the ureter exit.

URETERS AND URINARY BLADDER

The ureters extend inferiorly and medially from the renal pelvis at the renal hilum to reach the posterior and inferior surface of the urinary bladder which functions to store urine. The urinary bladder is a hollow muscular container that lies in the pelvic cavity just posterior to the symphysis pubis. In the male it is just anterior to the rectum; in the female it is just anterior to the vagina and inferior and anterior to the uterus. The ureters enter the bladder inferiorly on its posterolateral surface, and the urethra exits the bladder inferiorly and anteriorly.

At the junction of the urethra with the urinary bladder, smooth muscle of the bladder forms the internal urinary sphincter. The external urinary sphinctor is skeletal muscle that surrounds the urethra as the urethra extends through the pelvic floor. In the male the urethra extends to the end of the penis where it opens to the outside. The female urethra, about 4 cm long is much shorter and broader than the male urethra and opens into the vestibule anterior to the vaginal opening. Short wide urethra renders females more prone to urinary tract infections than male.

REGIONS OF THE ABDOMEN (FIGURE 6 – 1)

For descriptive purposes, the abdomen is divided into nine regions by the intersection of imaginary planes:

TWO HORIZONTAL PLANES

The upper horizontal plane (trans-pyloric) lies at the level of the first lumbar vertebra, midway between the suprasternal notch and the symphysis pubis

The lower horizontal plane passes through the upper borders of the iliac fossa

TWO SAGITTAL PLANES

Two lines drawn vertically from the mid-inguinal points towards the mid-clavicular points

Another system divides the abdomen into four regions by imaginary lines crossing at the umbilicus, forming the right upper, right lower, left upper, and left lower quadrants.

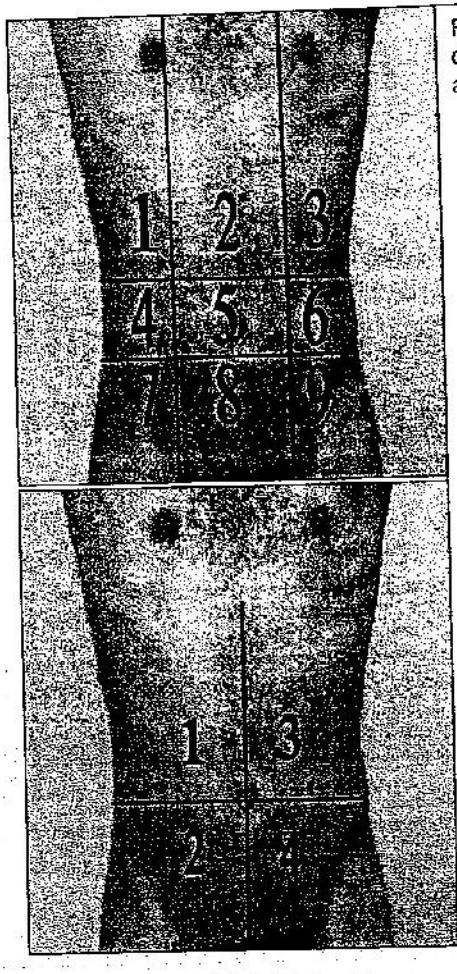


FIGURE 6 – 1. Regions of the abdomen are obtained either with division to nine regions or four regions. The nine regions of the abdomen include:

1. Right hypochondrium
2. Epigastric region
3. Left hypochondrium
4. Right flank region
5. Umbilical region
6. Left flank region
7. Right iliac fossa
8. Hypogastrium or suprapubic region
9. Left iliac fossa region

The four regions of the abdomen include:

1. Right upper quadrant
2. Right lower quadrant
3. Left upper quadrant
4. Left lower quadrant

EXAMINATION OF THE ABDOMEN

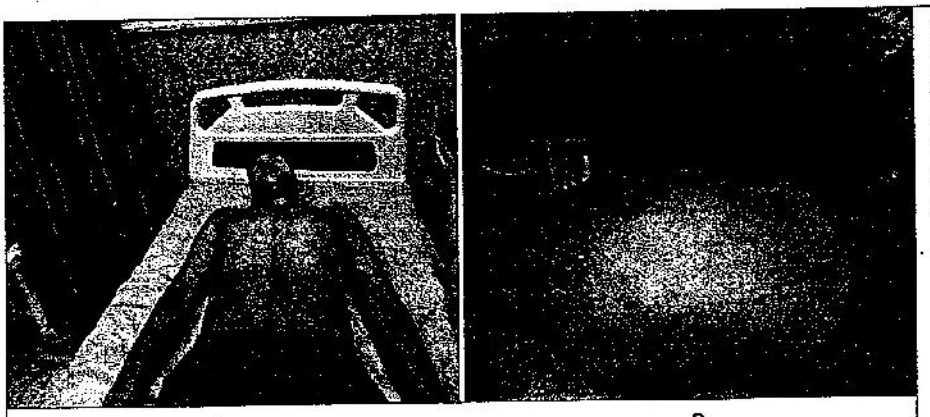
The patient should lie flat in bed and the abdomen should be fully exposed from the sternum to the knees. The genitalia should be covered with sheet or towel. The arms should be at the sides or folded across the chest and the legs flat. Frequently, patients tend to place their arms behind their head, which tightens the abdominal muscles and makes the examination more difficult. Placing a pillow beneath the knees often aids in relaxation. The candidate's hands and stethoscope should be warm (rub your hands), and nails short before examination. The abdominal examination is performed always on the right side and consists of inspection, palpation, percussion, and auscultation.

INSPECTION

At first stand at the end of the bed and inspect the whole abdomen (FIGURE 6 – 2); ask the patient to cough so as to elicit hernial expansile cough impulse. A hernia is a protrusion of an intra-abdominal structure through an abnormal opening; this may occur because of weakening of the abdominal wall by previous surgery (incisional hernia), a congenital abdominal wall defect, or chronically increased intra-abdominal pressure. Then sit or kneel on the right side of the couch so that the patient's abdomen is at eye level (FIGURE 6 – 2) watching for evidence of asymmetrical movement, indicating the presence of a mass. In particular a large liver may be seen to move below the right costal margin or a large spleen below the left costal margin. Inspection should include:

PATTERN OF MOVEMENT

The normal pattern of abdominal movement is a gentle rise in the abdominal wall during inspiration and fall during expiration. In male and children the pattern of movement is abdominothoracic (mainly abdominal) while in female it is thoracoabdominal (mainly thoracic). Diminished or absent abdominal movement is called (silent abdomen) that may suggest peritonitis. Visible peristalsis may be a normal finding in very thin elderly patient with lax abdominal muscle. Visible peristalsis propagated from left hypochondrium passing down to the suprapubic area then ascend to the epigastrium suggests gross gastric dilatation. From left to right hypochondrium, visible peristalsis is more suggestive of pyloric obstruction. Visible pulsation in the epigastrium may be a normal finding in thin or nervous individual or may be due to abdominal aortic aneurysm or pulsatile liver due to tricuspid reflux.

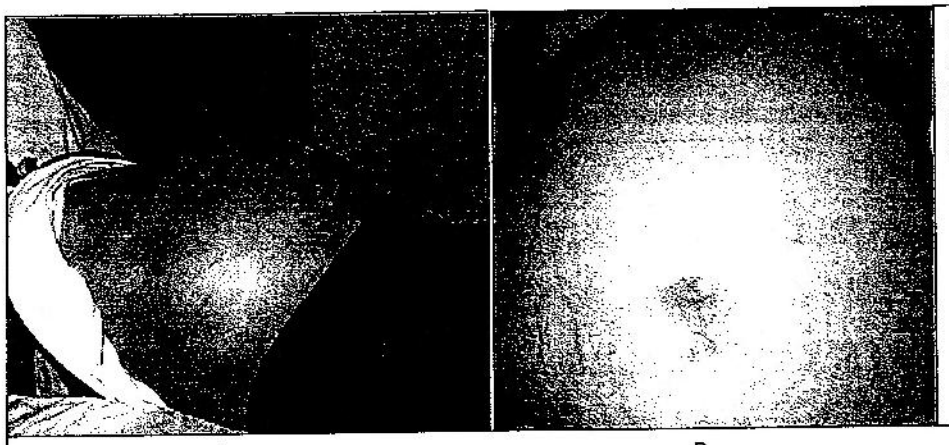


A B

FIGURE 6 – 2. Inspection of the abdomen from the end of the bed (A) and from the right side of the abdomen (B).

CONTOUR OF THE ABDOMEN

The abdomen normally has a gentle convex contour from side to side and from front to back with slightly retracted inverted umbilicus. A scaphoid or concave abdomen may be associated with advanced stage of starvation or malignancies e.g. stomach or esophagus. Abdominal distension (FIGURE 6 – 3) can be generalized and symmetrical with divercation of the skin (BOX 6 – 1) or local asymmetrical which may indicate enlargement of one of the abdominal or pelvic organs. Umbilicus normally lies at less equidistance between xiphysternum and symphysis pubis, is inverted, and had the same color of the rest of the abdomen. Downward displacement of the umbilicus suggests ascites while upward displacement occurs in pregnancy and ovarian cyst. In obesity, the umbilicus is usually sunken; in ascites, it is flat (transversely placed) or everted. Everted umbilicus also occurs in pregnancy. The Sister Joseph nodule is a metastatic tumour deposit in the umbilicus, the anatomical region where the peritoneum is closest to the skin. Discoloration of the umbilicus with a faintly bluish hue is found in retroperitoneal hemorrhage, abdominal aortic aneurysm rupture, and in hemorrhagic acute pancreatitis (Cullen's sign-the umbilical 'black eye').



A B

FIGURE 6 – 3. Abdominal distension (A) and Cullen's sign (B).

BOX 6 – 1. Causes of generalized symmetrical abdominal distention	SEVEN Fs
Fetus	Fat
Feces	Fibroid (tumor)
Flatus	Functional
Fluid	

TEXTURE OF THE ABDOMEN (FIGURE 6 – 4 and 6 – 5)

Normally the skin is shiny and smooth. Abnormal texture should be detected as well as its site according to the nine regions of the abdomen.

1 Tattoo

Tattoo can be a source for infection e.g. hepatitis B or hepatitis C if inadequately sterile needles are used. Tattoo is used for cosmetic or therapeutic purposes.

2 Spider naevi or spider angioma or spider telangiectasia

Spider telangiectasia is due to arteriolar changes and comprises a central arteriole (which occasionally raises the skin surface and blanches with pressure) from which small spider-like vessels radiate. They vary in size from 1-2 mm to 1-2 cm in diameter, are usually found only above the nipples, and can occur early in the disease. One or two small spider telangiectasia are found in about 2% of healthy people and can occur transiently in greater numbers in the third trimester of pregnancy, but otherwise they are a strong indicator of liver disease.

3 Linea nigra

The linea nigra is brownish black pigmented line extending from the umbilicus downwards in pregnancy

4 Erythema ab igne (thermal burn)

Erythema ab igne are brown mottled pigmentation. This type of erythema indicates application of hot water bottle or heat pad which is a sign of severe pain as in chronic pancreatitis

5 Campbell de Morgan

Campbell de Morgan is red spot that carries no medical significance.

6 Incisions, scars, and stoma

Color of the scar indicates its duration; white scar implies an old scar while red or pink is a recent scar. Stretched scar indicates incisional scar. Note also any surgical stomas

7 Striae

Stretching of the abdominal wall severe enough to cause rupture of the elastic fibres in the skin produces pink linear marks with a wrinkled appearance, which are called striae. When these are wide and purple-coloured, Cushing's syndrome may be the cause. Ascites, pregnancy or recent weight gain are much more common causes of striae

8 Abdominal hair distribution.

In male, the hair is distributed from pubis up ward to umbilicus in addition to its distribution on the chest down ward toward umbilicus (male escutcheon). In female, it is not uncommon for hair to spread from pubis up ward towards umbilicus but in lesser degree than of male. Presence of male distribution pattern in female is known as hirsutism

9 Prominent superficial veins

Below umbilicus, the normal venous flow in the abdominal wall is downwards. Above umbilicus, the normal venous flow in the abdominal wall is upwards. Normally usually only one or two veins (often epigastric) are visible. Prominent abdominal venous pattern with the direction of flow away from the umbilicus in all direction is often a reflection of portal hypertension and called caput Medusae (head of Medusa); venous collaterals with flow from the lower part of the abdomen toward the umbilicus suggest obstruction of the inferior vena cava; flow downward toward the umbilicus suggests superior vena cava obstruction.

If superficial veins are present, the direction of venous flow should be elicited at this stage. A finger is used to occlude the vein and blood is then emptied from the vein below the occluding finger with a second finger. The second finger is removed and, if the vein refills, flow is occurring towards the occluding finger. Flow should be tested separately in veins above and below the umbilicus.

10 Skin discoloration

Skin discoloration may also rarely occur in the flanks in severe cases of acute pancreatitis (Grey-Turner's sign)

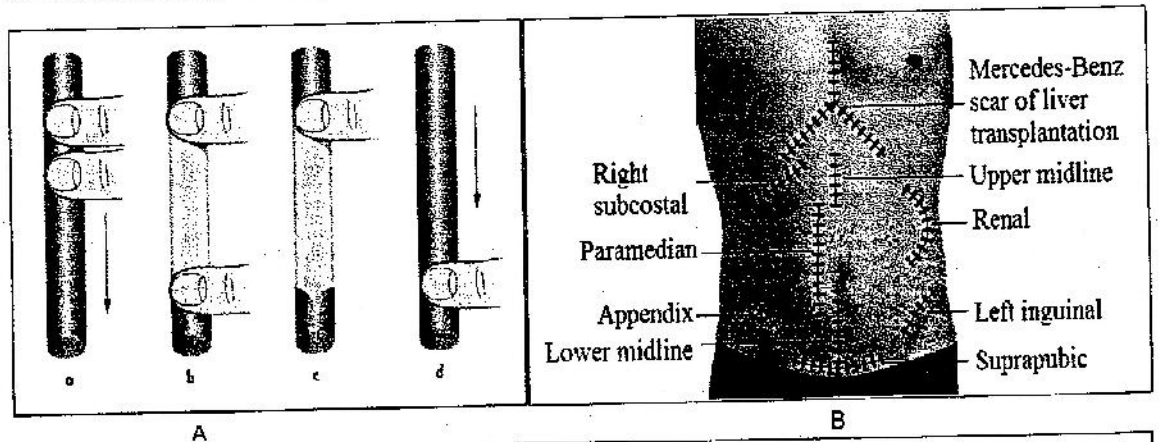


FIGURE 6 – 4. Detecting the direction of flow of a vein (A); (a) Place two fingers firmly on the vein. (b) The second finger is moved along the vein to empty it of blood and keep it occluded. (c) The second finger is removed but the vein does not refill. (d) At repeat testing and removing the first finger, filling occurs, indicating the direction of flow. Surgical scars or incisions (B).

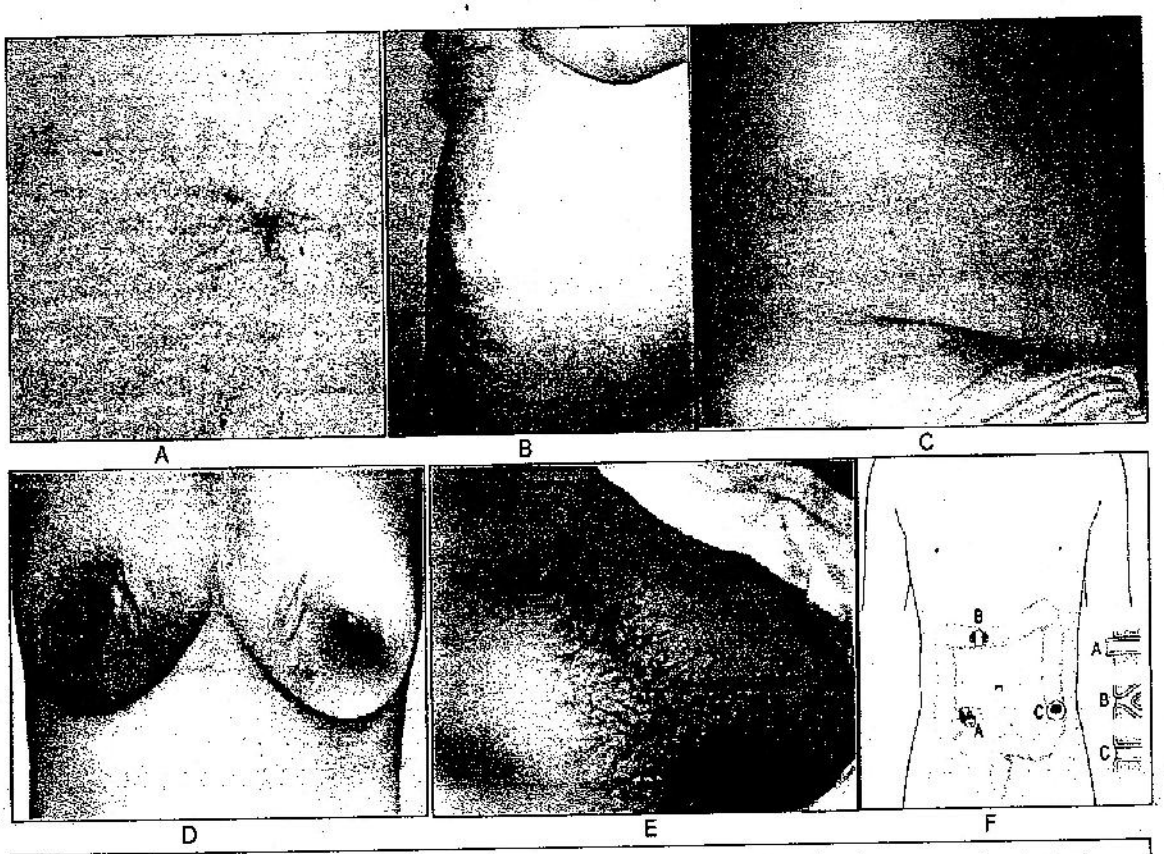


FIGURE 6 – 5. Spider angioma (A), caput medusa (B), gray turner sign (C), hirsutism in a women showing hair around the areola (D) and in the abdomen (E), and surgical stoma illustration where A refers to an ileostomy, B and C to colostomy.

PALPATION

The patient should lie in supine position during palpation. Always begin palpation in an area of the abdomen that is farthest from the location of pain. Palpation is performed under two ways; superficial (light or general) palpation and deep palpation. Before starting palpation, you should ask the patient about any abdominal pain and warm your hands (rub them). The abdominal muscles of the patient should be relaxed but if the patient is unable to relax try to:

1. Remove the pillow
2. Ask the patient to breathe deeply via the mouth
3. Bend the patient's knees up
4. Withdraw his/her attention

SUPERFICIAL (LIGHT OR GENERAL) PALPATION

Ask the patient to breathe from his/her mouth during palpation. If there is no abdominal pain, start from left iliac fossa; if present, start away from the site of pain in an anticlockwise direction. Place your right hand flat on the left iliac fossa with the wrist and forearm in the same horizontal plane then gentle with firm pressure with the fingers held almost straight with slight flexion at the metacarpophalangeal joint making an anticlockwise direction ending in suprapubic region. During the superficial palpation your eyes should be directed on the patient's face. The aims of superficial palpation are to detect superficial masses and superficial tenderness.

DEEP PALPATION

The aims of deep palpation are to detect deep masses, deep tenderness, and to palpate internal organs. Any mass must be carefully characterised and described (will be described later). Guarding of the abdomen (when resistance to palpation occurs due to contraction of the abdominal muscles) may result from tenderness or anxiety, and may be voluntary or involuntary. The latter suggests peritonitis. Rigidity is a constant involuntary contraction of the abdominal muscles always associated with tenderness and indicates peritoneal irritation. Rebound tenderness is said to be present when the abdominal wall, having been compressed slowly, is released rapidly and a sudden stab of pain results. This may make the patient wince, so the face should be watched while this maneuver is performed.

Palpation of internal organs usually includes palpation of the liver, spleen, both kidneys, abdominal aorta with its paraaortic lymph nodes, and lastly palpation of the common femoral vessels and inguinal lymph nodes.

PALPATION OF THE LIVER

The normal liver edge may be just palpable below the right costal margin on deep inspiration, especially in thin people. The normal upper border of the liver is level with the fifth intercostal space in the midclavicular line. The liver edge is then felt to be soft and regular with a fairly sharply defined border and the surface of the liver itself is smooth. The liver in health is non pulsatile and its span is 14 ± 2 cm.

FIRST METHOD (FIGURE 6 – 6)
Place your right hand transversely upon the right iliac fossa and ask the patient to take deep breath. With the height of inspiration, press your fingers firmly in wards upwards and move your hand parallel up until the costal margin. If the liver is enlarged, try to make out characters of the liver.

SECOND METHOD (FIGURE 6 – 6)
The right hand should be placed flat with the fingers pointing upwards and positioned so that the sensory fingers index and middle are lateral to the rectus muscle. The hand should be firmly pressed inwards and upwards with the height of inspiration. If the liver is enlarged, try to make out characters of the liver.

Detection of the lower edge of liver implies a palpable liver and not necessarily enlarged liver unless the liver span exceeds 14 – 16 cm. To estimate the liver span, follow the following points (6 – 6):

1. The upper edge is detected by percussion starting from the right second intercostal space downwards till percussion notes become dull which is normally at the fifth intercostal space
2. The lower edge of the liver is already detected by deep palpation. It is however advisable to start percussion from the right iliac fossa to upward toward the lower edge of the liver
3. The distance between the upper edge and the lower edge is detected and measured by tape measure

Other causes of a normal (normal liver span) but palpable liver include downward displacement due to emphysema, asthma or a subdiaphragmatic collection, or a Riedel's lobe. The Riedel's lobe is a tongue-like projection of the liver from the right lobe's inferior surface; it can be quite large and rarely extends as far as the right iliac fossa. It can be confused with an enlarged gallbladder or right kidney. Causes of enlarged liver are provided in BOX 6 – 2.

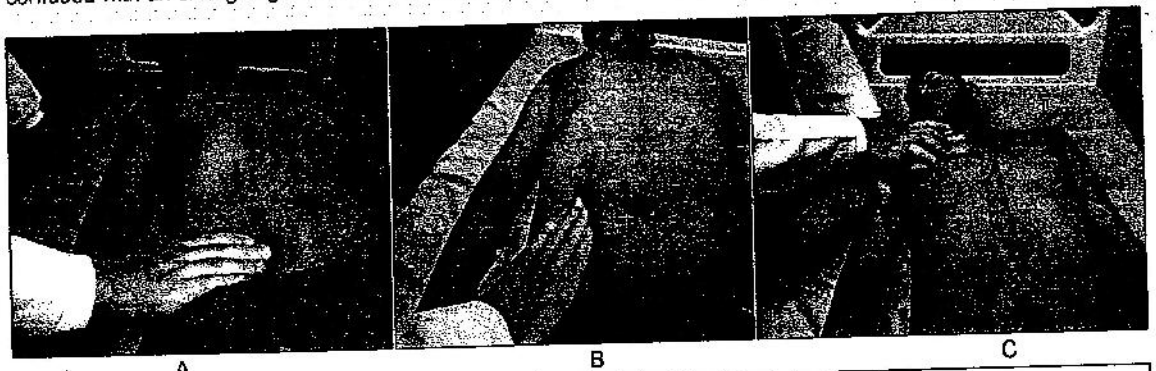


FIGURE 6 – 6. Two methods for liver palpation (A and B) and the technique of performing liver span (C).

WHAT ARE THE PHYSIOLOGIC CAUSES OF PALPABLE LIVER?
1. Normal thin individuals
2. Riedel's lobe

WHAT ARE THE CAUSES OF ENLARGED CIRRHOTIC LIVER?
1. Alcoholic liver disease
2. Hemochromatosis
3. Primary biliary cirrhosis
4. Development of hepatoma in patient with cirrhosis

BOX 6 – 2. Causes of enlarged liver

Chronic parenchymal liver disease	Malignancy	Right heart failure
Viral hepatitis Alcoholic liver disease Nonalcoholic steatohepatitis Hemochromatosis Autoimmune hepatitis Primary biliary cirrhosis	Primary hepatocellular cancer Secondary metastatic cancer	
	Other	Hematological disorders
	Congenital Riedel's lobe Amyloidosis Budd-Chiari syndrome Glycogen storage disorders	Lymphoma Leukaemia Myelofibrosis Polycythemia

Resonance below the 5th intercostal space suggests emphysema or occasionally the interposition of the transverse colon between the liver and the diaphragm (Chilaiditi's sign).

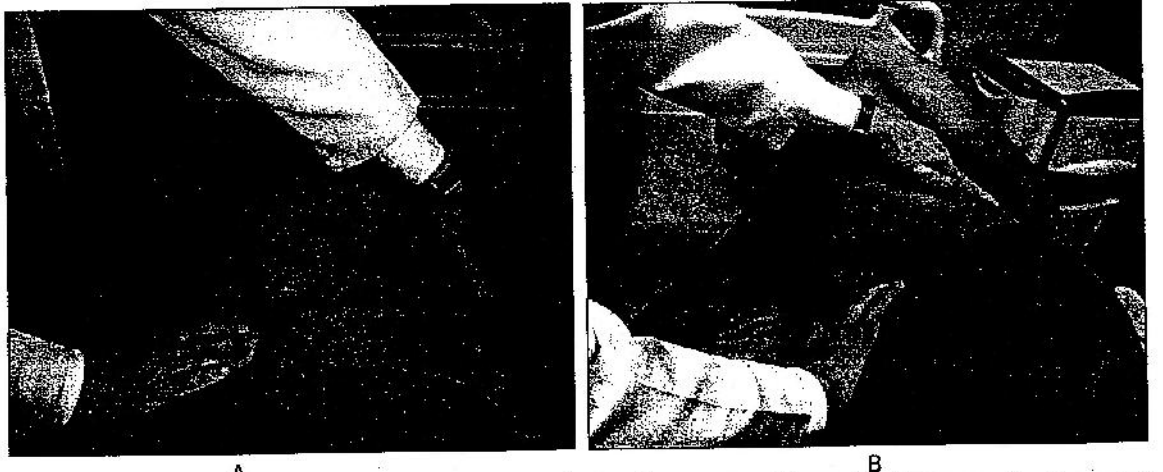
Palpation of the liver may result in carcinoid crises in patient with carcinoid syndrome. Patients will develop flushing, diarrhea, abdominal pain, cardiac abnormalities including tachycardia, hypertension, or hypotension.

CHARACTERISTICS OF ENLARGED LIVER; IF YOU FEEL A LIVER EDGE, DESCRIBE THE LIVER (SPECTS):

1	Size	The normal liver span is 14 ± 2 cm
Hepatomegaly means enlarged liver with span of more than 14 – 16 cm. If the liver span is less than 12 cm one may expect small liver e.g. cirrhosis		
2	Pulsatility	Pulsatile or not
Pulsatile liver suggests tricuspid regurgitation, hepatocellular cancer, or vascular abnormalities		
3	Edge	Regular or irregular
4	Consistency	Soft, firm, or hard
Firm and irregular liver may suggests hepatocellular carcinoma, metastatic disease, cirrhosis, hydatid disease, granuloma, and cysts		
5	Tenderness	Tender or not
Tender liver suggests hepatitis, biliary obstruction/cholangitis, right heart failure, Budd-Chiari syndrome, hepatic abscess, and hepatocellular cancer		
6	Surface	Smooth and regular or irregular

PALPATION OF THE SPLEEN

The normal spleen lies against the posterolateral wall of the abdominal cavity, beneath the ninth, tenth and eleventh ribs with its anterior border reaching the mid-axillary line. A normal spleen is not palpable. The spleen should be enlarged 2 – 3 times of its normal size so as to be palpable.



A B

FIGURE 6 – 7. Two methods for spleen palpation (A and B).

FIRST METHOD (FIGURE 6 – 7)
The left hand supports the tissues in the left lower costal area and the other hand is placed flat in the right iliac fossa. Ask the patient to take deep breath while your right hand is passed at the height of inspiration upward.

SECOND METHOD (FIGURE 6 – 7)
Turn the patient half his/her right side, your left hand supports the tissues in the left lower costal area and the right hand is placed on the left hypochondrium. Ask the patient to take deep breath whilst you palpate.

Main causes of splenomegaly are provided in BOX 6 – 3. Causes of hepatosplenomegaly are provided in BOX 6 – 4.

BOX 6 – 3. Causes of splenomegaly

Infection	Connective tissue diseases	Tumor	
Infectious mononucleosis	Still's disease	Angioma	
Typhoid fever	Felty's syndrome	Primary fibrosarcoma	
Tuberculosis	Metabolic	Hematological disorders	
Infective endocarditis	Rickets	Lymphoma	Hereditary spherocytosis
Malaria	Amyloidosis	Leukaemia	Auto immune hemolytic anemia
Schistosomiasis	Porphyria	Myelofibrosis	Thalassemia
Kala azar		Polycythemia rubra vera	

Massive (huge) splenomegaly is defined as enlarged spleen more than 8 cm below the costal margin and/or its drained weight is more than 1 kg. Some causes of massive (huge) splenomegaly are:

1. Chronic myeloid leukaemia
2. Visceral leishmaniasis (Kala azar)
3. Malaria
4. Myelofibrosis

Splenic ruptures have been reported to occur in infectious mononucleosis, cytomegalovirus infections, acute and chronic myelogenous leukemia, acute and chronic lymphocytic leukemia, myeloproliferative diseases, and non-Hodgkin lymphoma.

BOX 6 – 4. Causes of hepatosplenomegaly

Viral hepatitis	Myelofibrosis	Liver cirrhosis due to alcohol or hemochromatosis.
Infectious mononucleosis	Polycythemia rubra vera	Amyloidosis
Leukemia	Lymphoma	

BALLOTTEMENT TECHNIQUE
When significant ascites is present, abdominal viscera like liver or spleen or other masses may be difficult to feel by usual deep palpation. Here is the opportunity to practise dipping or ballottement technique. Using the hand placed flat on the abdomen, the fingers are flexed at the metacarpophalangeal joints rapidly so as to displace the underlying fluid. This enables the fingers briefly touch the surface of the structure covered in ascitic fluid through the abdominal wall. In particular, this should be attempted to palpate an enlarged liver or spleen (FIGURE 6 – 8). The liver and spleen may become ballottable when gross ascites is present.

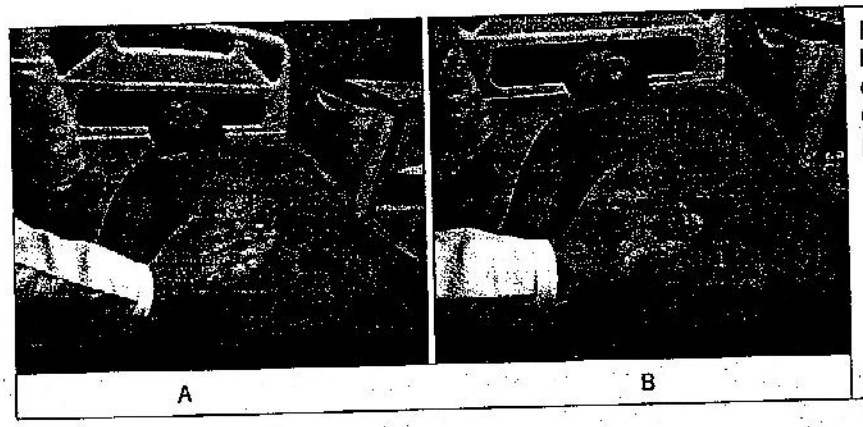


FIGURE 6 – 8. The ballottement technique for detection of abdominal organs or masses in the precence of ascites.

WHAT ARE THE CAUSES OF SPLENOMEGALY IN SICKLERS?

3. Sequestration crises
4. Splenic vein thrombosis
5. Sickle thalassemia syndrome

WHAT ARE THE CAUSES OF SPLENOMEGALY IN HEART FAILURE?

5. Infective endocarditis
6. Constrictive pericarditis
7. Amyloidosis
8. Cardiac cirrhosis

PALPATION OF BOTH KIDNEYS

More often, neither kidney can be palpated in the adult. Palpation of the right kidney is performed by deep palpation below the right costal margin whilst the patient is asked to deeply breathe. The candidate stands at the right side and places the left hand behind the patient's right flank, between the costal margin and the iliac crest. The right hand is placed just below the costal margin with the tips of the fingers pointing to the candidate's left (FIGURE 6 – 9). Very deep palpation may reveal the lower pole of the right kidney as it descends during inspiration. The lower pole may be felt as a smooth, rounded mass. The same procedure is used for the left kidney. Because the left kidney is more superior to the right, the lower pole of a normal left kidney is rarely palpable. Occasionally, the spleen may be mistaken for an enlarged left kidney. To differentiate spleen from left kidney:

1 Spleen moves downwards on inspiration and laterally then cross the midline
2 Spleen is not bimanually palpable but the kidney is bimanually palpable
3 On percussion; dull percussion notes suggest spleen while resonant percussion notes suggest kidney as it covered by colon
4 In splenomegaly you can not get above it while in left kidney you can
5 Splenic notch can be felt in lower medial border

FIGURE 6 – 9. The techniques for right kidney (A) and left kidney (B) palpation.

Causes of enlarged kidneys are provided in BOX 6 – 5.

BOX 6 – 5. Causes of enlarged kidney

Hydronephrosis	Tumour of the kidney	Polycystic kidney
Pyonephrosis	Solitary cyst	Renal hypertrophy
Perinephric abscess		

PALPATION OF THE ABDOMINAL AORTA AND PARAAORTIC LYMPH NODES

Abdominal aorta is detected by deep palpation a little above and to the left of the umbilicus by two hands held side by side deeply into the abdominal wall to note its pulsation (FIGURE 6 – 10). In thin patient, the aorta is more easily palpable. Arterial pulsation from the abdominal aorta may be present, usually in the epigastrium, in thin normal people. The problem is to determine whether such a pulsation represents an aortic aneurysm (usually due to atherosclerosis) or not. Measure the width of the pulsation gently with two fingers by aligning these parallel to the aorta and placing them at the outermost palpable margins. With an aortic aneurysm, the pulsation is expansile (i.e. it enlarges appreciably with systole) (FIGURE 6 – 11). If an abdominal aortic aneurysm is larger than 5 cm in diameter, it usually merits surgical repair. Paraaortic lymph nodes are lying along the left border of the aorta and become palpable only when they are enlarged.

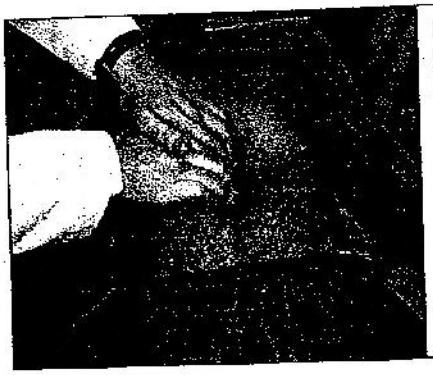


FIGURE 6 – 10. The techniques for abdominal aorta and paraaortic lymph nodes palpation. Paraaortic lymph nodes are lying along the left border of the aorta and become palpable only when they are enlarged.

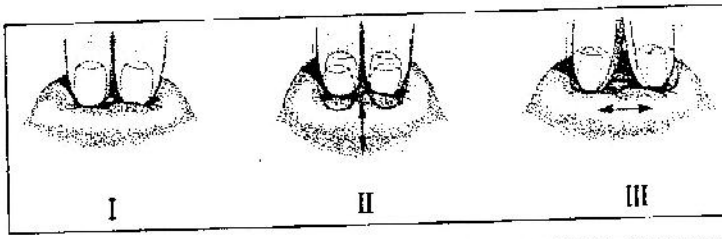


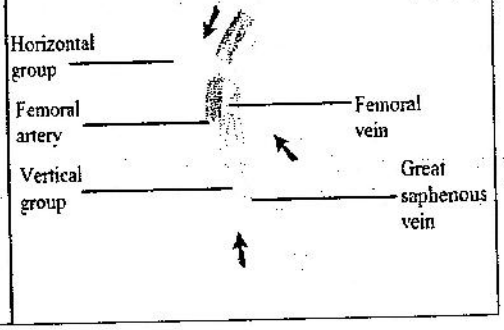
FIGURE 6 – 11. Detecting an expansile impulse where in I = no impulse; in II = transmitted pulsation from a neighboring artery; and in III = expansile impulse, the sign of an aneurysm.

PALPATION OF THE COMMON FEMORAL VESSELS AND THE INGUINAL LYMPH NODES

The common femoral vessels are found just below the inguinal ligament at the mid-point between anterior superior iliac spine and symphysis pubis (FIGURE 6 – 12). Palpation is done by placing the pulps of the right index, middle and ring fingers over this site in the groin then compare with the contralateral femoral vessels.

Inguinal lymph nodes are divided into horizontal group as well as lateral and medial vertical group (FIGURE 6 – 12). The horizontal group drains from the lower abdomen below umbilicus. The medial vertical group drains from the lower limb, from skin of penis, scrotum, perineum, lower vagina, vulva and anus. The lateral vertical group drains from buttock and back. Start palpation firstly over the horizontal chain, which just lies below the inguinal ligament and then over the vertical chain along the saphenous veins.

FIGURE 6 – 12. Anatomy of the inguinal area showing the femoral vessels and the inguinal lymph nodes.



PERCUSSION

Undergraduate and postgraduate students should routinely percuss the liver, the spleen, the urinary bladder, and the abdomen to detect ascites or gaseous distension.

PERCUSSION OF THE LIVER

The liver borders should be percussed routinely to determine the liver span. If the liver edge is not palpable and there is no ascites, the right side of the abdomen should be percussed from the right iliac fossa up to the right costal margin until dullness is encountered. This defines the liver's lower border even when it is not palpable. The upper border of the liver must always be defined by percussing down the midclavicular line starting from the right second intercostal space. As the chest is percussed downward, the resonant note of the chest becomes dull as the liver is reached. As percussion continues, this dull note becomes tympanic because the percussion is now over the colon. Loss of normal liver dullness may occur in massive hepatic necrosis, or with free gas in the peritoneal cavity (e.g. perforated bowel).

PERCUSSION OF THE SPLEEN (FIGURE 6 – 13)

Percussion for splenic dullness is accomplished with any of three techniques:

1 Nixon's method For postgraduate students

The patient is placed on the right side so that the spleen lies above the colon and stomach. Percussion begins at the lower level of pulmonary resonance in the posterior axillary line and proceeds diagonally along a perpendicular line

toward the lower midanterior costal margin. The upper border of dullness is normally 6–8 cm above the costal margin. Dullness >8 cm in an adult is presumed to indicate splenic enlargement.

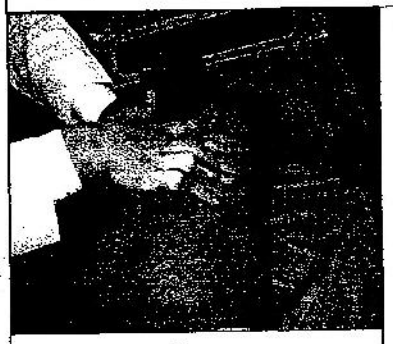
2 Castell's method For undergraduate students and postgraduate students

With the patient supine, percussion in the lowest intercostal space in the anterior axillary line (8th or 9th) produces a resonant note if the spleen is normal in size. This is true during expiration or full inspiration. A dull percussion note on full inspiration suggests splenomegaly.

3 Percussion of Traube's semilunar space For postgraduate students

The borders of Traube's space are the sixth rib superiorly, the left midaxillary line laterally, and the left costal margin inferiorly. The patient is supine with the left arm slightly abducted. During normal breathing, this space is percussed from medial to lateral margins, yielding a normal resonant sound. A dull percussion note suggests splenomegaly.

A B C



D

FIGURE 6 – 13. Percussion of the lower border of the liver (A), and the techniques for splenic percussion including Nixon method (B), Castell's method (C), and Traube's method (D).

PERCUSSION OF URINARY BLADDER

The candidate should start percussion from the umbilicus downward to the suprapubic area (FIGURE 6 – 14). The suprapubic area when percussed may sound dull if the urinary bladder is distended, if the uterus is enlarged, or in the presence of pelvic mass.

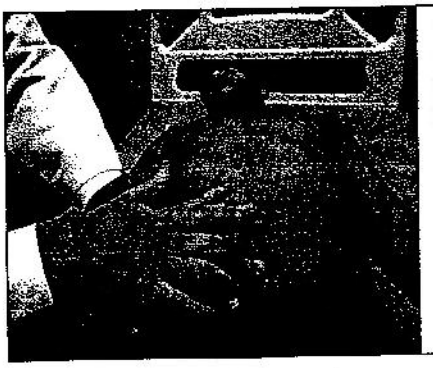


FIGURE 6 – 14. Percussion of the urinary bladder.

PERCUSSION OF THE ABDOMEN

Routine abdominal examination should include percussion starting in the midline with the finger pointing towards the patient's face; the percussion note is tested out towards the flanks on each side. The percussion note over most of the abdomen is resonant, due to air in the intestines. The resonance is detectable out to the flanks. When peritoneal fluid (ascites) collects, the influence of gravity causes this to accumulate first in the flanks in a supine patient. Thus, a relatively early sign of ascites is a dull percussion note in the flanks. With gross ascites, the abdomen distends, the flanks bulge, umbilical eversion occurs and dullness is detectable closer to the midline. However, an area of central resonance will always persist. Two methods are used to detect ascites:

1 SHIFTING DULLNESS (FIGURE 6 – 15)

To detect this sign, while standing on the right side of the bed percuss out to the left flank until dullness is reached. This point should be marked and the patient rolled towards the candidate. Ideally 30 seconds to 1 minute should then pass so that fluid can move inside the abdominal cavity and then percussion is repeated over the marked point. Shifting dullness is present if the area of dullness has changed to become resonant. This is because peritoneal fluid moves under the influence of gravity to the right side of the abdomen when this is the lowermost point. Then the patient is asked to return to supine position and repeat the procedure, but to the contralateral side. Positive shifting dullness indicates collection of fluid of about one liter. False positive shifting dullness occurs in large intra-abdominal ovarian cyst (BOX 6 – 6) and when loops of the intestine contain an excessive amount of fluid.

2 FLUID OR TRANSMITTING THRILL (FIGURE 6 – 15)

To detect a fluid thrill the candidate asks an assistant (or the patient) to place the medial edge of his/her palm firmly on the centre of the abdomen so as to dampen any impulse that may be transmitted through the fat of the abdominal wall. The candidate then flicks the side of the abdominal wall, and a pulsation (thrill) is felt by the hand placed on the other abdominal wall. As a rule, transmitting thrill is felt only when there is a large amount of ascites present under tension. Interestingly it may also occur when there is a massive ovarian cyst or a pregnancy with hydramnios.

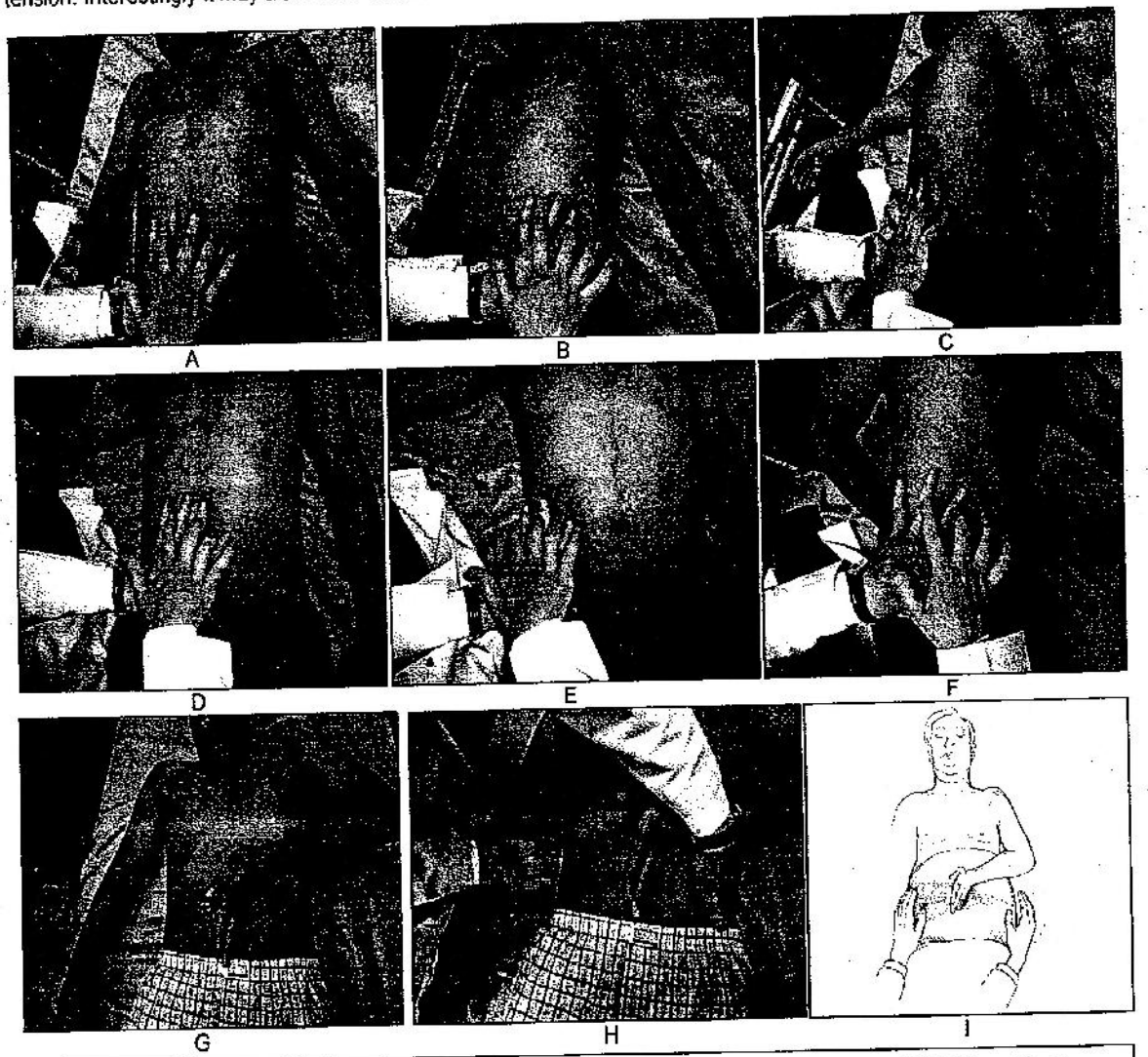


FIGURE 6 – 15. The techniques for performing shifting dullness (A – F) and transmitting thrill (G, H, and I). The shifting dullness method comprises:

A) Percussion starting in the midline
B) Percussion is continued to the left flank first.
C) Ask the patient to turn toward you and repeat flank percussion after 30 – 60 seconds from turning
D) Ask the patient to lie supine again and percuss from flank to the midline.
E) Repeat the percussion from the midline to the right flank area
F) Ask the patient to turn to opposite side and percuss the right flank area after 30 – 60 seconds.

BOX 6 – 6. Distinction between gross ascites and large ovarian cyst	
Gross ascites	Large ovarian cyst
The umbilicus is displaced downwards	The umbilicus is displaced upwards
Shifting dullness and fluid thrill are positive	Fluid thrill is only positive in the front, not in flanks
Percussion notes: Central: resonant (tympanic) Flanks: dullness	Percussion notes: Central: dullness Flanks: resonant (tympanic)
Distention is mainly central	Distension is felt arising from the pelvis that one cannot get above the swelling

Ascites can be classified according to the amount of protein in the ascetic fluid (BOX 6 – 7) into exudative ascites and transudative ascites. Cirrhotic patients typically develop transudative ascites with a total protein concentration below 25 g/l. However, in up to 30% of patients, the total protein concentration is more than 30 g/l. In these cases it is useful to calculate the serum – ascites albumin gradient (SAAG) by subtracting the concentration of the ascites fluid albumin from the serum albumin. A gradient of more than 11 g/l (or > 1.1 g/dl) is strongly suggestive of portal hypertension and cirrhosis.

BOX 6 – 7. Common causes of ascites	
Exudate > 25 g/l of protein	Transudate < 25 g/l of protein
Peritoneal carcinomatosis Pancreatic ascites Infection (pneumococcal or tuberculosis) Budd-Chiari syndrome	Hepatic cirrhosis (portal hypertension) Congestive heart failure Nephrotic syndrome Ovarian tumour (Meig's syndrome)

Chylous ascites refers to a turbid, milky, or creamy peritoneal fluid due to the presence of thoracic or intestinal lymph with triglyceride concentration of >200 mg/dl. A turbid fluid due to leukocytes or tumor cells may be confused with chylous fluid (pseudochylous), and it is often helpful to carry out alkalinisation and ether extraction of the specimen. Alkali tends to dissolve cellular proteins and thereby reduce turbidity; ether extraction leads to clearing if the turbidity of the fluid is due to lipid. Rarely, ascitic fluid may be mucinous in character, suggesting either pseudomyxoma peritonei or rarely a colloid carcinoma of the stomach or colon with peritoneal implants.

CLINICAL CLUE

The diagnosis of chylous ascites requires the presence of > 200 mg/dl of triglyceride in the peritoneal fluid. The diagnosis of chylous pleural effusion requires the presence of > 110 mg/dl of triglyceride in the pleural fluid.

AUSCULTATION

With the patient lying in supine position, place the stethoscope diaphragm over the abdomen to listen for (FIGURE 6 – 16) bowel sounds, arterial bruits, venous hum, and succussion splash in the following sequence:

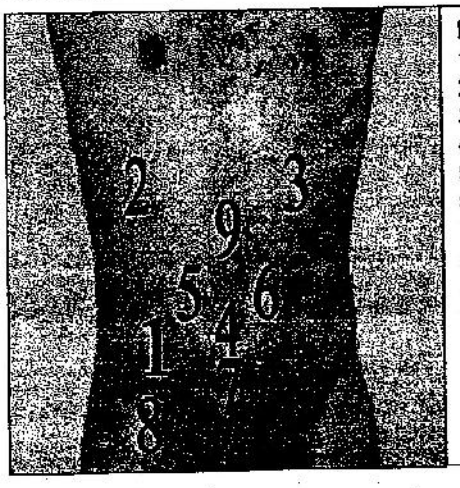


FIGURE 6 – 16. The sites of abdominal auscultation include:
1 Mc Burney's point for bowel sounds
2 Right hypochondrium for hepatic bruits and friction rub
3 Left hypochondrium for splenic bruits and friction rub
4 Over the umbilicus for venous hum of portal hypertension
5 and 6 Over both renal arteries for arterial bruits in renal artery stenosis
7 Above or below the umbilicus for arterial bruits of atherosclerotic aorta
8 Over the femoral arteries for arterial bruits
9 Over the epigastrium for succussion splash

1 BOWEL SOUNDS

Mc Burney's point, lies at the junction of the lateral one third and the medial two third of a line joining the anterior superior iliac spine and the umbilicus, is used to listen for bowel sounds. Normal bowel sounds occur intermittently every 5 – 10 seconds and have a soft gurgling character. Bowel sounds are described as either present or absent when after 2 minutes no bowel sounds are heard. The absence of bowel sounds suggests a paralytic ileus or peritonitis. The presence of normal bowel sounds makes obstruction unlikely. The bowel that is obstructed produces a louder and higher-pitched sound with a tinkling quality due to the presence of air and liquid ('obstructed bowel sounds'). There may be rushes of low-pitched rumbling sounds, termed borborygmi, which are associated with hyperperistalsis. They are common in early acute intestinal obstruction, hunger patients, severe gastrointestinal bleeding, small bowel malabsorption, and carcinoid tumour.

2 OVER THE RIGHT HYPOCHONDRIUM

At the right hypochondrium (the liver site), listen for hepatic arterial bruits and friction rub. Hepatic arterial systolic bruit is higher pitched than a venous hum, is not continuous and is well localised. This is usually due to a hepatocellular cancer but may occur in acute alcoholic hepatitis or with an arteriovenous malformation. A friction rub, which sounds like rubbing your dry fingers together, is a rough creaking or grating noise heard as the patient breathes and may be heard over the liver (perihepatitis) due to hepatic tumour (hepatocellular cancer or metastases) or hepatic infection (liver abscess, gonococcal or chlamydial perihepatitis).

3 OVER THE LEFT HYPOCHONDRIUM

At the left hypochondrium (the spleen site), listen for splenic friction rub that suggests perisplenitis and splenic bruits that suggests arteriovenous fistula

4 OVER THE UMBILICUS (KENAWY'S SIGN)

A venous hum is a continuous, low-pitched, soft murmur that may become louder with inspiration and diminish when more pressure is applied to the stethoscope. Typically it is heard over the umbilicus in cases of portal hypertension.

5 AND 6 OVER BOTH RENAL ARTERIES

Renal arteries are located approximately 2 – 3 cm on either side of the midline above the umbilicus. Listen at these sites for renal artery bruits which indicate renal artery stenosis

7 ABOVE OR BELOW THE UMBILICUS

Listen above the umbilicus over the aorta for arterial bruits, which suggest an atheromatous or aneurysmal aorta or superior mesenteric artery stenosis and listen below the umbilicus at the bifurcation of the abdominal aorta for arterial bruits which also suggest atherosclerosis

8 OVER THE FEMORAL VESSELS

At the common femoral vessels, listen for arterial bruits that suggest atherosclerosis process and increase the probability for renal artery stenosis if present. Listen also for pistol shot that occurs in aortic valve regurgitation.

9 SUCCUSSION SPLASH (FIGURE 6 – 17)

A succussion splash sounds like a half-filled water bottle being shaken. Explain the procedure to the patient and ask him/her about the time of last meal. To elicit a succession splash, place the stethoscope at the epigastrium and your hands over the lower ribs (some time over the pelvis) and shake the patient quickly and rhythmically from side to side. An audible splash more than 4 hours after the patient has eaten or drunk anything indicates delayed gastric emptying, e.g. pyloric stenosis.

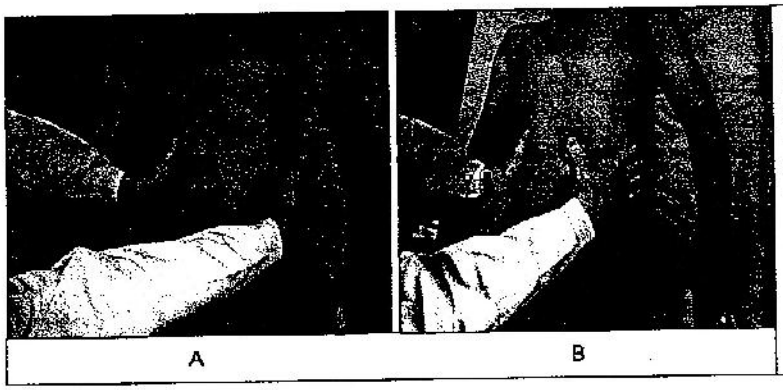


FIGURE 6 – 17. The technique of succussion splash including shaking the patient quickly and rhythmically from side to side firstly without stethoscope (A) and secondly with stethoscope (B).

When the candidate had reached to this point, he/she should request from the examiners to perform the following examination:

1. Renal angle tenderness
2. Examination of the genitalia
3. Digital rectal examination
4. Examination of hernia

RENAL ANGLE TEST (MURPHY'S KIDNEY PUNCH)

You may note tenderness when examining the abdomen, search for it at each costovertebral angle. The costovertebral angle—the angle formed by the lower border of the 12th rib and the transverse processes of the upper lumbar vertebrae—defines the region to assess for kidney tenderness (FIGURE 6 – 18). Pressure from your fingertips may be enough to elicit tenderness, but if not, use fist percussion. This is performed by striking the costovertebral angle with the ulnar surface of your fist. Use enough force to cause a perceptible but painless jar or thud in a normal person. These techniques are shown in FIGURE 6 – 18.

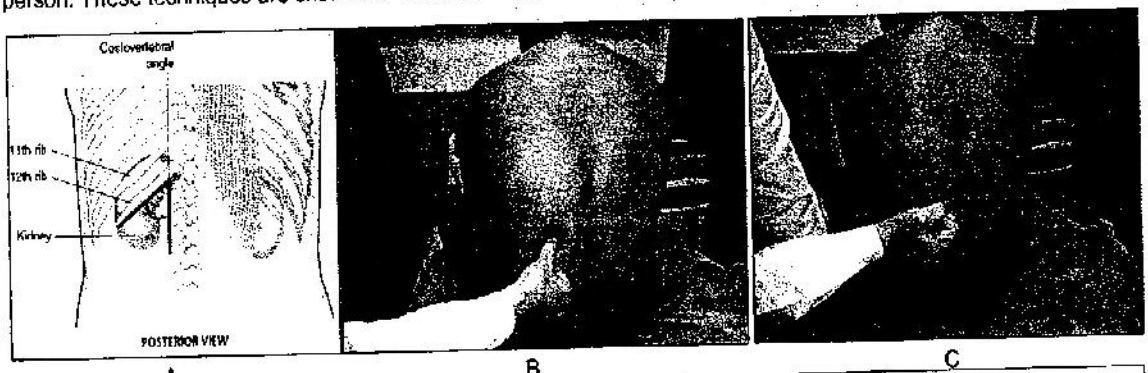


FIGURE 6 – 18. The anatomy of the renal angle (A) and the techniques of renal angle tenderness. Pressure from your fingertips may be enough to elicit tenderness (B), but if not, use fist percussion (C).

EXAMINATION OF MALE GENITALIA

The candidate should wear gloves for this part of examination. Examination of the male genitalia is performed with the patient first lying on the couch and then standing. This postural change is important, because hernias or scrotal mass may not be apparent in the lying position. Anatomy of male genitalia is shown in FIGURE 6 – 19.

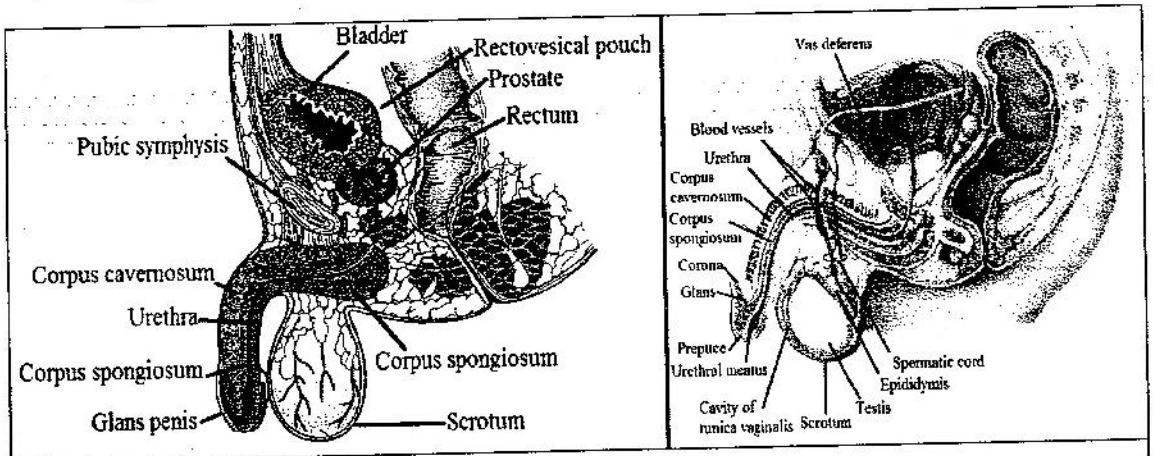


FIGURE 6 – 19. Anatomy of the external genitalia.

ANATOMY OF MALE GENITALIA

The shaft of the penis is formed by three columns of vascular erectile tissue: the corpus spongiosum, containing the urethra, and two corpora cavernosa. The corpus spongiosum forms the bulb of the penis, ending in the cone-shaped glans with its expanded base, or corona. In uncircumsized men, the glans is covered by a loose, hood-like fold of skin called the prepuce or foreskin. The urethra is located ventrally in the shaft of the penis; urethral abnormalities may sometimes be felt there. The urethra opens into the vertical, slit-like urethral meatus, located somewhat ventrally at the tip of the glans. The scrotum is a loose, wrinkled pouch divided into two compartments, each of which contains a testis or testicle. The testes are ovoid, somewhat rubbery structures, about 4.5 cm long in the adult, with a range from 3.5 cm to 5.5 cm. The left usually lies somewhat lower than the right. On the posterolateral surface of each testis is the softer, comma-shaped epididymis. It is most prominent along the superior margin of the testis. The epididymis may be located anteriorly in 6% to 7% of males. Surrounding the testis, except posteriorly, is the tunica vaginalis, a serous membrane enclosing a potential cavity. The vas deferens, a cord-like structure, begins at the tail of the epididymis, ascends within the scrotal sac, and passes through the external inguinal ring on its way to the abdomen

and pelvis. Behind the bladder it is joined by the duct from the seminal vesicle and enters the urethra within the prostate gland. Sperm thus pass from the testis and the epididymis through the vas deferens into the urethra. Secretions from the vasa deferentia, the seminal vesicles, and the prostate all contribute to the semen. Within the scrotum each vas is closely associated with blood vessels, nerves, and muscle fibers. These structures make up the spermatic cord.

EXAMINATION TECHNIQUE

Inspect the genitals for congenital anomalies including hypospadius and epispadius, and mucosal lesion (BOX 6 – 8).

BOX 6 – 8. Causes of genital lesions

Ulcerative	Non-ulcerative
Herpes simplex (vesicles followed by ulcers: tender) Syphilis (non-tender) Malignancy (squamous cell carcinoma: non-tender) Chancroid (Hemophilus ducreyi infection: tender) Behçet's syndrome Granuloma inguinale (Klebsiella granulomatis)	Balanitis, due to Reiter's syndrome or poor hygiene Venereal warts Primary skin disease, e.g. psoriasis

Look also for urethral discharge; compress the glans gently between your index finger above and your thumb below. This maneuver should open the urethral meatus and allow you to inspect it for discharge. Normally there is none. Profuse yellow discharge may be seen in gonococcal urethritis; scanty white or clear discharge in nongonococcal urethritis. If the patient has reported a discharge but you do not see any, ask him to strip, or milk, the shaft of the penis from its base to the glans. Alternatively, you can do so.

Palpate any abnormality of the penis between your thumb and first two fingers, noting any tenderness or induration. Induration along the ventral surface of the penis suggests a urethral stricture or possibly a carcinoma. Tenderness of such an indurated area suggests periurethral inflammation secondary to a urethral stricture.

Inspect the scrotum with the patient lying at first and then standing. Usually the left testis hangs lower than the right. This is the only part of the body that consistently does not appear bilaterally symmetrical on inspection. Torsion of the testis may cause the involved testis to appear higher and to lie more transversely than normal. Inspect for edema of the skin, sebaceous cysts, tinea cruris or scabies. Scrotal edema is common in severe cardiac failure and may occur with the nephrotic syndrome and ascites. Palpate each testis gently using the fingers and thumb of the right hand. The testes are normally equal in size, smooth and relatively firm. Absence of one or both testes may be due to previous excision, failure of the testis to descend, or a retractile testis. In children the testis may retract as examination of the scrotum begins because of a marked cremasteric reflex. A maldescended testis (one that lies permanently in the inguinal canal or higher) has a high chance of developing malignancy. An exquisitely tender, indurated testis suggests orchitis. This is often due to mumps in postpubertal patients and occurs about 5 days after the parotitis. An undescended testis may be palpable in the inguinal canal, usually at or above the external inguinal ring. The presence of small firm testes suggests an endocrine disease (hypogonadism) or testicular atrophy due to alcohol or drug ingestion. Feel posteriorly for the epididymis and then upwards for the vas deferens and the spermatic cord. It should be possible to differentiate the vas from the testis.

Assess any scrotal swelling as a lump (BOX 6 – 9 and FIGURE 6 – 20).

BOX 6 – 9. Differential diagnoses of scrotal swelling

Painless scrotal swelling		Painful scrotal swelling
Tumour Chronic epididymoorchitis e.g. T.B. Inguinal hernia Chronic hematocele Varicocele	Hydrocele Scrotal edema Spermatocele Epididymal cyst	Torsion of the testis Acute epididymoorchitis Strangulated inguinal hernia Acute hematocele

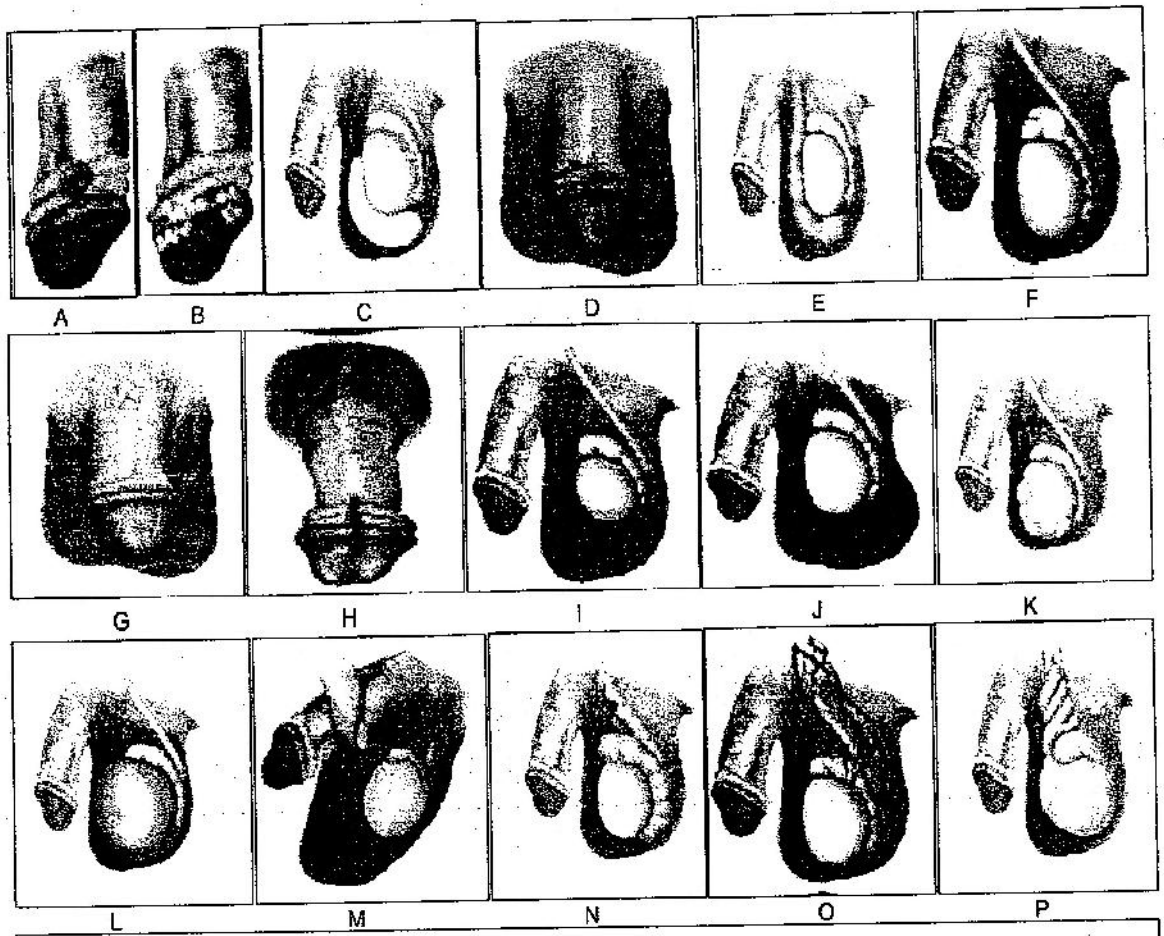


FIGURE 6 – 20. Clinical signs in male genitalia including:

(A) A syphilitic chancre appears as a non-tender oval or round, dark red, painless erosion or ulcer.

(B) Venereal warts (Condyloma acuminatum) are rapidly growing excrescences that are moist and malodorous. They result from infection by human papillomavirus.

(C) A hydrocele is a non-tender, fluid-filled mass within the tunica vaginalis. It transilluminates and the examining fingers can get above the mass within the scrotum.

(D) Genital herpes appears as cluster of small vesicles, followed by shallow, painful, red based ulcers.

(E) Scrotal hernia within the scrotum so it is usually an indirect inguinal hernia.

(F) Spermatocele and cyst of the epididymis. A painless, movable cystic mass just above the testis suggests a spermatocele or an epididymal cyst. Both transilluminate. The former contains sperm and the latter does not, but they are clinically indistinguishable.

(G) Peyronie's disease that consists of palpable non-tender hard plaques just beneath the skin, usually along the dorsum of the penis. The patient complains of crooked, painful erections.

(H) Hypospadias is a congenital displacement of the urethral meatus to the inferior surface of the penis.

(I) Small testis; small firm testes occur in Klinefelter's syndrome while small soft testes suggesting atrophy seen in cirrhosis, mytonic dystrophy, use of estrogens, hypopituitarism; may also follow orchitis.

(J) Scrotal edema.

(K) Tumor of the testis.

(L) Acute orchitis that may be difficult to distinguish from the epididymis.

(M) Cryptorchidism. The testis is atrophied and may lie in the inguinal canal or the abdomen, resulting in an undeveloped scrotum. Cryptorchidism markedly raises the risk of testicular cancer.

(N) Tuberculous epididymitis produces a firm enlargement of the epididymis, which is sometimes tender, with thickening or beading of the vas deferens.

(O) Varicocele refers to varicose veins of the spermatic cord, usually found on the left. It feels like a soft "bag of worms" separate from the testis, and slowly collapses when the scrotum is elevated in the supine patient. Infertility may be associated.

(P) Torsion, or twisting, of the testicle on its spermatic cord produces an acutely painful, tender, and swollen organ that is retracted upward in the scrotum. The scrotum becomes red and edematous. There is no associated urinary infection. Torsion, most common in adolescents, is a surgical emergency because of obstructed circulation.

EXAMINATION OF THE ANUS AND RECTUM

Place the patient in left lateral decubitus with the buttocks at the edge of the couch, the knees drawn up to the chest and the heels clear of the perineum; gloves should be worn. Separate the buttocks and inspect the perianal and anal region for any one of the following conditions (FIGURE 6 – 21):

1 Thrombosed external hemorrhoids (piles)

Small (less than 1 cm), tense bluish swellings may be seen on one side of the anal margin. They are painful and are due to rupture of a vein in the external hemorrhoidal plexus. Hemorrhoids are not palpable unless thrombosed

2 Skin tags

Skin tags are fleshy, pedunculated masses. They are composed of hyperplastic epidermis over a fibrous connective tissue stalk. These can be an incidental finding or occur with hemorrhoids or Crohn's disease

3 Rectal prolapse

Circumferential folds of red mucosa are visible protruding from the anus. These may become apparent only when the patient is asked to strain as if to pass stool

4 Anal fissure (fissure-in-ano)

This is a crack in the anal wall that usually occur directly posteriorly and in the midline. A tag of skin may be present at the base: this is called a sentinel pile. It indicates that the fissure is chronic. It may be necessary to get the patient to bear down for a fissure to become visible. Multiple or broad-based fissures may be present in patients with inflammatory bowel disease, malignancy or venereal disease

5 Fistula-in-ano

It is a hole or dimple that its entrance may be visible, usually within 4 cm of the anus. The mouth has a red pouting appearance caused by granulation tissue. This may occur with Crohn's disease or perianal abscess

6 Condylomata acuminata (anal warts)

These warts are caused by human papillomavirus. These may be confused with skin tags, but are in fact pedunculated papillomas with a white surface and red base. They may surround the anus

7 Carcinoma of the anus.

This may be visible as a fungating mass at the anal verge

8 Pruritus ani

The appearance of this irritating anal condition varies from weeping red dermatitis to a thickened white skin

9 Excoriation from diarrhoea

10 Pilonidal sinuses

Pilonidal infections and chronic pilonidal sinuses typically occur in the midline of the sacrococcygeal skin of young men as a hole or dimple existing in the midline of natal cleft posterior to anus. Hair seems to play a central role in the process of infection and in the perpetuation of granulation tissue in sinuses. This is consistent with the clinical observation that pilonidal patients are hirsute and that pilonidal disease rarely occurs in patient with less body hair

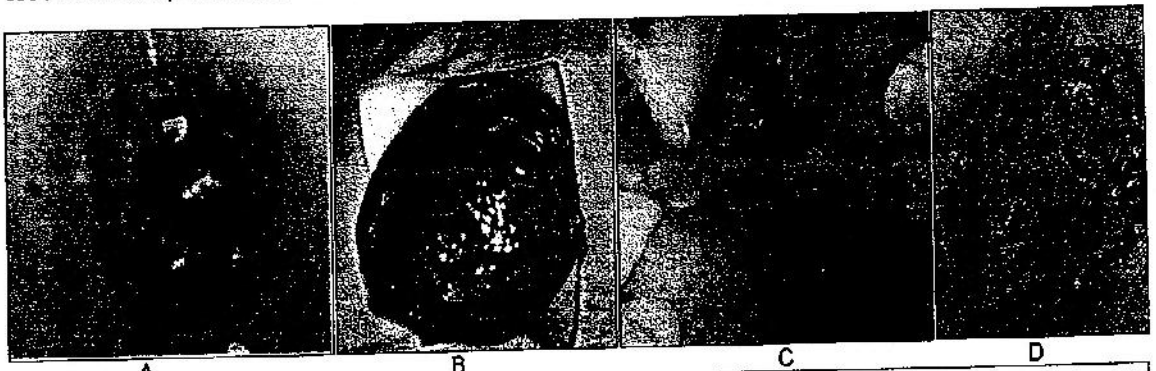


FIGURE 6 – 21. Anal conditions including piles (A), rectal prolapse (B), anal fissure (C), and anal warts (D).

DIGITAL RECTAL EXAMINATION (DRE)

This is an important part of the examination and should not be avoided simply because it is considered unpleasant. Before you begin examination explain to the patient what you are going to do and ask for chaperone. The position of during digital rectal examination may involve one of the following two positions:

1 The modified lithotomy position (patient on his back with knees flexed)

This position is used when the patient has difficulty turning or when a detailed examination of the anus is not required. The candidate passes the right hand under the patient's right thigh. The index finger in the patient's rectum is used in conjunction with the candidate's left hand, which is placed on the abdomen. This bimanual approach is useful and causes minimal disturbance of the sick patient

2 Sims' position (the left lateral prone position)

This is the position that is commonly used

The steps of examination should follow the ABCDEF rules:

A ALARM THE PATIENT

The candidate should tell the patient that a lubricant that will feel cool will be used, and this will be followed by the sensation of having to move the bowels; the patient should be assured that he will not do so.

B BREATHE DEEPLY

The candidate lubricates the right gloved index finger and places the left hand on the patient's buttocks. As the left hand spreads the patient's buttocks, the candidate's right index finger is gently placed on the anal verge. The sphincter should be relaxed by gentle pressure with the palmar surface of the finger. The patient is asked to take a deep breath, at which time the right index finger is inserted into the anal canal as the anal sphincter relaxes.

C COUGH

The sphincter tone should be assessed. The sphincter should close completely around the examining digit. Ask the patient now to squeeze the examining finger or to cough to assess the tone of the anal musculature.

D DIGITAL ROTATION

The finger should be inserted as far as possible into the rectum, although 10 cm is the probable limit of digital exploration. Rotate the finger through 360° in the canal and feel for any irregularity of the wall

E EXAMINATION

Assess the rectal wall in relation to its anterior wall where the anatomy from below upward is that: membranous urethra, the prostate and base of the bladder. The prostate should be assessed in the form of size, shape, consistency and tenderness. Normally the prostate is 2.5 cm in length, about the size of chestnut, regular, and smooth with firm rubbery consistency, and having two lateral regular lobes and median sulcus. In prostatic carcinoma, it becomes enlarged with irregular surface and hard consistency as well as irregular nodular lateral lobes and distortion of the median sulcus. In benign prostatic hyperplasia, the prostate becomes enlarged and soft in consistency, not hard.

F FINGER WITHDRAWAL

Finally, withdraw your finger and look at it for evidence of pus, mucus or blood.

SPECIAL CLINICAL TECHNIQUES AND SIGNS

ACUTE APPENDICITIS

Peritoneal irritation can be elicited on physical examination by the findings of voluntary and involuntary guarding, percussion, or rebound tenderness (Blumberg's sign). Any movement, including coughing (Dunphy's sign), may cause increased pain. Other findings may include sever tenderness when the Mc Burney point is pressed with the index finger (Mc Burney sign); pain in the right lower quadrant during palpation of the left lower quadrant (Rovsing's sign), pain on internal rotation of the hip (obturator sign, suggesting a pelvic appendix), and pain on extension of the right hip (iliopsoas sign, typical of a retrocecal appendix).

ACUTE HEMORRHAGIC PANCREATITIS

Cullen's sign (a faint bluish discoloration around the umbilicus indicative of hemoperitoneum) or Grey Turner's sign (a blue-red or green-brown discoloration of the flanks reflecting tissue catabolism of hemoglobin) may be seen.

GASTRIC CARCINOMA

Lymph node involvement in the left supraclavicular area is termed Virchow's node and periumbilical nodal involvement is called Sister Mary Joseph's node. Metastasis to the ovary is termed Krukenberg's tumor. Paraneoplastic syndromes may precede or occur concurrently with gastric cancer. Examples include Trousseau's syndrome (recurrent migratory superficial thrombophlebitis), acanthosis nigricans, and neuromyopathy.

SPLENIC RUPTURE

Pain at the tip of the left shoulder which is referred pain due to irritation of the undersurface of the left leaf of the diaphragm that has the same nerve supply of the shoulder (Kehr sign) and presence of shifting dullness in the right flank area while in the left flank, it is constant dullness due to presence of blood and clots (Balance's sign).

ACUTE CHOLECYSTITIS

Murphy's sign or Moynihan's sign involves placing of the thumb of the left hand over a point between xiphesternum and the flank and the remaining fingers over the costal area. The patient is then asked to take a deep breath; the inspiration is sharply arrested due to the bump of the gall bladder against your thumb and it is a diagnostic sign for acute cholecystitis.

FIGURE 6 – 21. Eliciting Murphy's sign.

CLINICAL CLUE

Gall bladder is enlarged due to obstruction of the cystic duct usually by gall stone (the patient here is not jaundiced) or obstruction of the common bile duct (CBD) usually by gall stones or carcinoma of the head of the pancreas (the patient here is jaundiced). Courvoisier's law stated that a palpable gall bladder in presence of painless obstructive jaundice suggests carcinoma of the head of the pancreas.

Drawbacks of Courvoisier's sign

1. Obese patient may have impalpable gall bladder even when enlarged.
2. Double impaction; one at the cystic duct and the other at the common bile duct.
3. Klatskin tumour (tumors arising at the bifurcation of the common bile duct).

EXAMPLES

Examination of the abdomen had shown normally moving abdomen with gentle rise during inspiration and descent during expiration; limited visible area of epigastric pulsation, normal contour with the umbilicus being placed at less equidistance between xiphisternum and symphysis pubis, and normal abdominal wall texture with no scars, incision, or stomas. No superficial or deep tenderness or mass; impalpable liver with a span of 12 cm; impalpable spleen with negative Castell's sign and impalpable kidneys; no lymphadenopathy. Percussion notes were resonant over all the abdomen; no suprapubic dullness. Bowel sounds were present with no additional bruits, hum, or friction rub. Examination of the genitalia had shown normally appearing genitalia with no obvious, swelling, tenderness, or masses; normally appearing anus and normal digital rectal examination with good sphincter tone; prostate had regular smooth surface, firm in consistency, normal lateral lobes and median sulcus. No blood, pus, or mucus, were seen.

What is the interpretation?
The interpretation is normal abdominal examination

Examination of the abdomen had shown normally moving abdomen with gentle rise during inspiration and descent during expiration as well as limited visible area of epigastric pulsation. Asymmetrical visible abdominal swelling in the left hypochondrium region. The umbilicus being was placed at less equidistance between xiphisternum and symphysis pubis. Normal abdominal wall texture with no scars, incision, or stomas. No superficial tenderness; impalpable liver with a span of 12 cm; enlarged spleen for 10 cm below the costal margin with positive Castell's sign and impalpable kidneys; no lymphadenopathy. Percussion notes were resonant over all the abdomen apart from the left hypochondrium region where the percussion notes where dull, no suprapubic dullness. Bowel sounds were present with no additional bruits, hum, or friction rub. Examination of the genitalia had shown normally appearing genitalia with no obvious, swelling, tenderness, or masses; normally appearing anus and normal digital rectal examination with good sphincter tone; prostate had regular smooth surface, firm in consistency, normal lateral lobes and median sulcus. No blood, pus, or mucus, were seen.

What is the interpretation?
The interpretation is huge splenomegaly

Examination of the abdomen had shown generalised symmetrical distention of the abdomen, limited visible area of epigastric pulsation, and no visible peristalsis. The umbilicus was everted and displaced down ward. There where small piece of surgical dressing applied on the abdominal wall in the right iliac fossa, no visible superficial veins, and no other superficial scars or stomas. No superficial or deep tenderness or mass; impalpable liver with a span of 7 cm; the spleen was not palpable by deep palpation but palpable with ballottement method with positive Castell's sign; impalpable kidneys; no lymphadenopathy. There were positive shifting dullness and transmitting thrills. Bowel sounds were present with no additional bruits, hum, or friction rub. Examination of the genitalia had shown normally appearing genitalia with no obvious, swelling, tenderness, or masses; normally appearing anus and normal digital rectal examination with good sphincter tone; prostate had regular smooth surface, firm in consistency, normal lateral lobes and median sulcus. No blood, pus, or mucus, were seen.

What is the interpretation?
The interpretation is portal hypertension probably due to liver disease

Examination of the abdomen had shown normally moving abdomen with gentle rise during inspiration and descent during expiration; limited visible area of epigastric pulsation, normal contour with the umbilicus being placed at less equidistance between xiphisternum and symphysis pubis, and normal abdominal wall texture with no scars, incision, or stomas. There was right hypochondrial tenderness; the liver was enlarged with regular and smooth edge and surface; soft consistency; tender but non-pulsatile, with a span of 15 cm; palpable spleen to 1 cm below the left costal margin with positive Castell's sign and impalpable kidneys; no lymphadenopathy. Percussion notes were resonant over all the abdomen; no suprapubic dullness. Bowel sounds were present with no additional bruits, hum, or friction rub. Examination of the genitalia had shown normally appearing genitalia with no obvious swelling, tenderness, or masses; normally appearing anus and normal digital rectal examination with good sphincter tone; prostate had regular smooth surface, firm in consistency, normal lateral lobes and median sulcus. No blood, pus, or mucus, were seen.

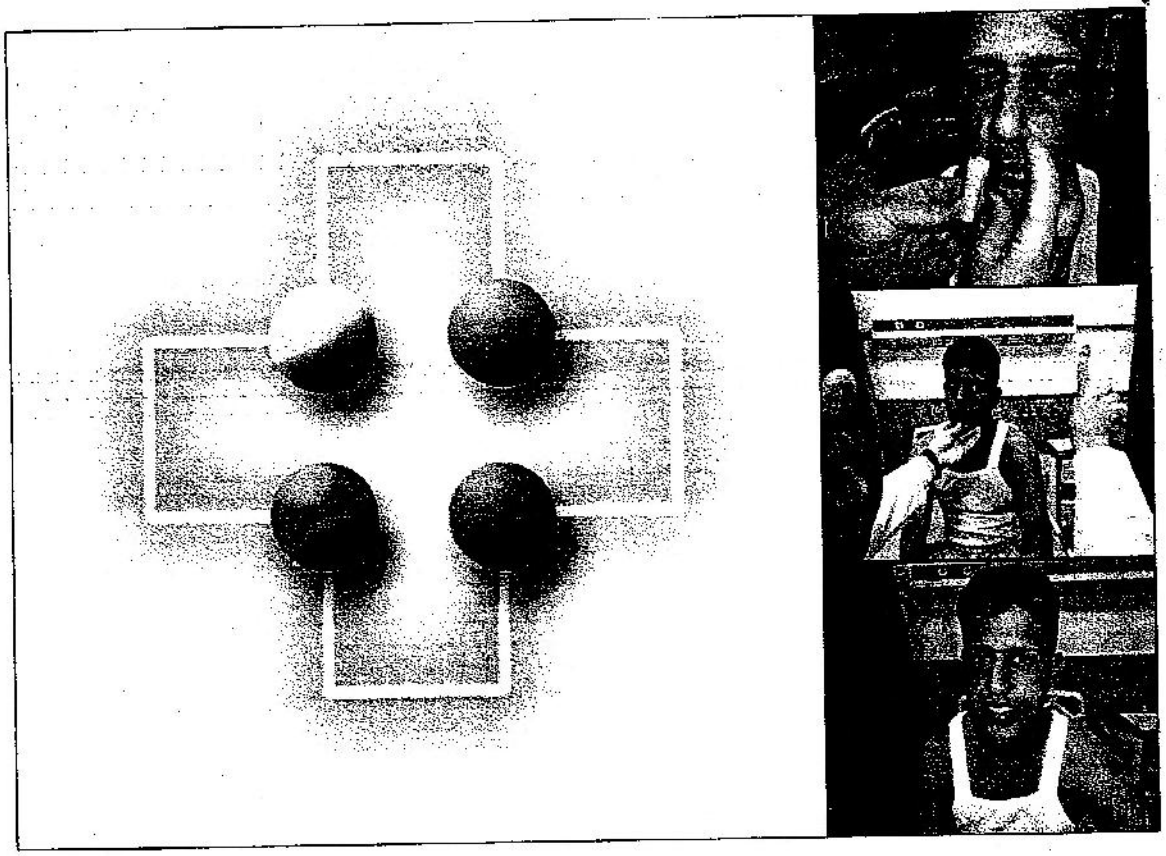
What is the interpretation?
The interpretation is hepatosplenomegaly (hepatitis??)

Further reading

- Hutchison's clinical methods
- Davidson's principles and practice of medicine
- Harrison's principles of internal medicine
- Cecil principles of internal medicine
- Macleod's clinical examination
- Oxford Handbook of Clinical Examination – Practical Skills
- First Aid for the Internal Medicine Boards
- Clinical Examination - A Systematic Guide to Physical Diagnosis
- Textbook of Clinical Neurology
- Queen Square Textbook of Neurology
- Dictionary of Neurological Signs
- Clinical Neurology (McGraw-Hill, 2009)
- 2005 Adams and Victor's Principles of Neurology
- Bailly and Love's short practice of surgery
- Gynecology by ten teachers
- Obstetrics by ten teachers
- Hamilton Bailly's physical signs in clinical surgery
- Browse; signs and symptoms of surgical diseases
- Nelson's text book of pediatrics
- Bate's guide to physical diagnosis.
- Textbook of physical diagnosis.
- Oxford handbook of clinical examination/ practical skills.
- Kochar's Clinical Medicine for Students.

7

NERVOUS SYSTEM EXAMINATION



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ANATOMY OF THE NERVOUS SYSTEM

The cells of the nervous system are divided into two types. These are:

Neuron or nerve cell which consists of three parts. These parts are

1. Neuron cell body or nerve cell body or soma
2. Dendrites which are short highly branched cytoplasmic extensions. Functionally, they conduct action potentials toward cell body
3. Axon. Most neurons have single axon that arises from axon hillock. Axon may remain single or form collateral axons. Functionally, axon conducts action potentials to the dendrites, cell bodies or axons of other neurons

Non neuronal cells (neuroglia) which are existing in five types

1. Astrocytes that participate in blood–brain barrier (BBB)
2. Ependymal cells that line the ventricular cavities of the brain and central canal of the spinal cord
3. Microglial cells that have phagocytic activity
4. Oligodendrocytes that form myelin sheaths around axons in the central nervous system (CNS)
5. Schwann cells that form myelin sheaths around axons in the peripheral nervous system (PNS)

DIVISIONS OF THE NERVOUS SYSTEM

The nervous system consists of the CNS that includes the brain and spinal cord and PNS that is composed of nerves and ganglia. PNS is subdivided into sensory (afferent) division which transmits action potentials from the sensory organs into the CNS and motor (efferent) division which transmits action potentials from the CNS into the effector organs e.g. muscles and glands. This motor division is further divided into somatic nervous system that transmits action potentials from CNS to the skeletal muscles and autonomic nervous system which transmits action potentials from CNS to the smooth, and cardiac muscles and certain glands.
Autonomic nervous system is also further divided into sympathetic nervous system which prepares the body for physical activities if activated and parasympathetic nervous system which regulates resting or vegetative functions e.g. digestion and voiding.

ORGANIZATION OF THE NERVOUS SYSTEM

Bundles of parallel axons with their sheaths are whitish in colour and called white matter. In the CNS, they form the nerve tracts which transmit action potentials from one area of the CNS to another. In the PNS, bundles of axons form the nerves. Collection of nerve cell bodies and their associated dendrites are gray in color and called gray matter. In CNS, they comprise cerebral and cerebellar cortex and red nuclei. Red nuclei include the basal ganglia, which affect movement, and the thalamus and the hypothalamus (structures in the diencephalon). Out side CNS, collection of nerve cell bodies are called ganglia.

THE BRAIN

The brain has four regions: the cerebrum, the diencephalon, the brainstem, and the cerebellum.

THE CEREBRUM

It is the largest portion of the brain. The cerebrum is divided into left and right hemispheres by a longitudinal fissure. The most conspicuous features on the surface of each hemisphere are numerous folds called gyri. Between which, there are intervening grooves called sulci. Each cerebral hemisphere is divided into four lobes. The frontal lobe is important in voluntary motor function, motivation, mood and aggression. The parietal lobe is the major centre for reception and evaluation of most sensory information (excluding smell, hearing and vision). The frontal and parietal lobes are separated by central sulcus. The occipital lobe functions in the reception and integration of the visual input. The temporal lobe receives and evaluates olfactory and auditory (hearing) input and plays an important role in memory. The temporal and the parietal lobes are separated by lateral sulcus.
The gray matter of the cerebrum on the outer surface is the cortex and deeply inside the cerebrum are the red nuclei. The white matter of the cerebrum is located between cerebral cortex and the nuclei and called cerebral medulla which constitutes the nerve tracts that connect the cerebral cortex to other areas of the cortex or other parts of the CNS. These tracts fall into three main categories:

1. Association fibers connect areas of the cerebral cortex within the same hemisphere
2. Commissural fibers connect one cerebral hemisphere to the other
3. Projection fibers connect between the cerebrum and other parts of the brain and spinal cord. The majority of these form the internal capsule (FIGURE 7 – 1). This structure consists of an anterior limb, a posterior limb, and a segment between them, the knee.

The cortex of the right cerebral hemisphere controls muscular activity in, and receives sensory input from, the left half of the body. The left cerebral hemisphere controls muscular activity in, and receives sensory input from, the right half of the body. Sensory information received by the cortex of one hemisphere is shared with the other through the commissural fibers.

THE BASAL GANGLIA

Several basal ganglia are located next to the genu of the internal capsule (FIGURE 7 – 1), e. g., the globus pallidus and the putamen. The globus pallidus and the putamen together are also known as the lentiform nucleus. The anterior limb of the internal capsule is bounded by the caudate nucleus and the lentiform nucleus, which together form the corpus striatum. The basal ganglia have an important function in the so-called extrapyramidal motor system.

Putamen and globus pallidus=lentiform body.
Caudate nucleus and lentiform body=corpus striatum.

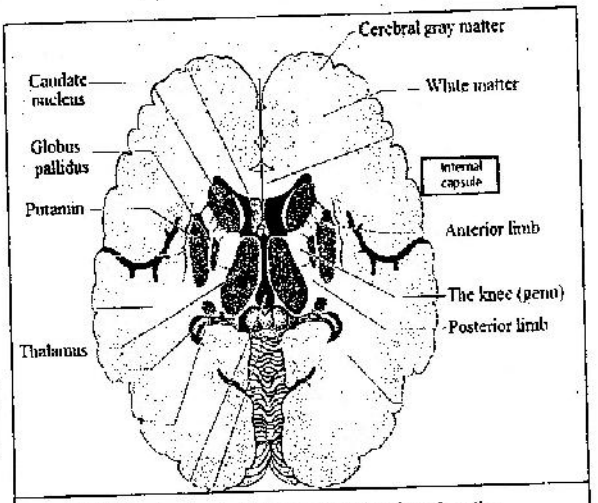


FIGURE 7 – 1. Anatomy of the brain showing the internal capsule and the basal ganglia

DIENCEPHALON

Diencephalon (FIGURE 7 – 2) is an area of the brain that lies between cerebral hemispheres and surrounds third ventricle. It consists of the thalamus, which is the major subcortical relay station for all incoming impulses except those dealing with smell (the olfactory pathway). In the thalamus, these impulses are synaptically transmitted to neurons that mostly project to the cerebral cortex. The hypothalamus is an area essential to life. It regulates body temperature, manages water balance, controls food intake, governs emotional life, and regulates the autonomic nervous system. Through the hypophysial stalk (infundibulum), the diencephalon becomes the most important connection to the chief organ of hormonal regulation, the pituitary gland. The diencephalon also includes the medial and lateral geniculate bodies, important relay stations in the visual and auditory pathways subsumed under the term metathalamus, the pineal body (pineal gland), and the habenular nuclei forming the epithalamus.

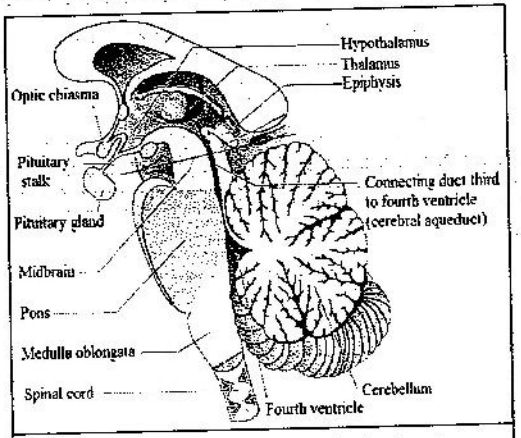


FIGURE 7 – 2. Anatomy of the brain showing the parts of the diencephalon.

THE BRAIN STEM (FIGURE 7 – 2)

The midbrain, pons, and medulla oblongata form the brainstem that extends from base of brain to foramen magnum. The nuclei of the cranial nerves lie in the brainstem. Cranial nerves I and II are actually fiber tracts emerging from the brain. Medulla oblongata contains cranial nerves XII, XI, X, and IX; the pons contains cranial nerves VIII, VII, VI, and V; the midbrain contains cranial nerves IV and III.

I. MIDBRAIN

The midbrain is the smallest part of the brain, lying between the diencephalon and the pons. Several ascending and descending tracts run through the midbrain.

II. PONS

The pons lies between the midbrain and the medulla oblongata and is separated from the cerebellum above it by a cavity, the fourth ventricle. Several ascending and descending tracts run through the pons.

III. MEDULLA OBLONGATA

The posterior surface of the medulla is partly covered by the cerebellum. Anteriorly it has a median groove (median sulcus), interrupted by the decussation of the pyramidal tracts. On each side of the groove the medulla has two raised areas (nucleus cuneatus and nucleus gracilis); relay station in the ascending sensory tracts of the posterior spinal funiculus (fasciculi cuneatus and gracilis). The medulla, like the pons and the midbrain, contains ascending and descending fibers and nuclei of cranial nerves. It is also the seat of the respiratory and circulatory centers.

THE CEREBELLUM

The cerebellum is organized like cerebrum with gray matter both on outside as cortex and on inside as nuclei. The cerebellar cortex has gyri and sulci, but the gyri are much smaller than those of the cerebrum. The cerebellum consists of three portions; the flocculonodular lobe, a narrow central vermis, and two large lateral hemispheres. The flocculonodular lobe involves in balance, the anterior portion of the vermis is involved in gross motor coordination and the posterior vermis together with the large lateral hemispheres are involved in fine motor coordination.

THE SPINAL CORD

The spinal cord is a cylindrical mass of nerve tissue encased within the bony vertebral column, extending from the medulla to the first or second lumbar vertebra. It contains important motor and sensory nerve pathways that exit and enter the cord via anterior and posterior nerve roots and spinal and peripheral nerves. The spinal cord also mediates reflex activity of the deep tendon (or spinal nerve) reflexes. A cross section of the spinal cord shows that the cord consists of a central gray matter and a peripheral white portion (FIGURE 7 – 3).

The white matter consists of nerve tracts and the gray matter consists of nerve cell bodies and dendrites. The white matter in each half of spinal cord is organized into three columns called the anterior (ventral), posterior (dorsal) and lateral columns. Each column is subdivided into tracts. Individual axons carrying impulses to and from the brain are usually grouped together within the tract. The central gray matter is organized into horns. Each half of the central gray matter of the spinal cord consists of a relatively thin posterior (dorsal/sensory) horn and a large anterior (ventral/motor) horn. Small lateral horn also exists in levels of the cord associated with the autonomic nervous system. Dorsal and ventral roots exit the spinal cord near the dorsal and ventral horns. The dorsal root is formed by axons of the sensory neuron and also contains dorsal root ganglia. The ventral root is formed by the axon of neurons in the anterior and lateral horns. The dorsal and ventral roots unite to form the spinal nerves. There are 31 pairs of spinal nerves, each having a ventral (motor) and dorsal (sensory) root. The ventral root consists of efferent nerve fibers, which originate in the anterior and lateral (T1-T2 only) gray matter and travel to the peripheral nerve and muscle. This is the motor root. The dorsal root consists of afferent nerve fibers whose cell bodies are in the dorsal root ganglion. This is the sensory root. The spinal nerves are grouped into 8 cervical (C1-C8), 12 thoracic (T1-T12), 5 lumbar (L1-L5), 5 sacral (S1-S5), and 1 coccygeal nerve (FIGURE 7 – 3).

Note that the spinal cord is not as long as the vertebral canal. The level of the nerve roots exiting the cord differs from the adjacent vertebral level. The lumbar and sacral roots travel the longest intraspinal distance. These roots fan out like a horse's tail at L1–2, giving rise to the term cauda equina. To avoid injury to the cord, most lumbar punctures are performed at the L3–4 vertebral interspace.

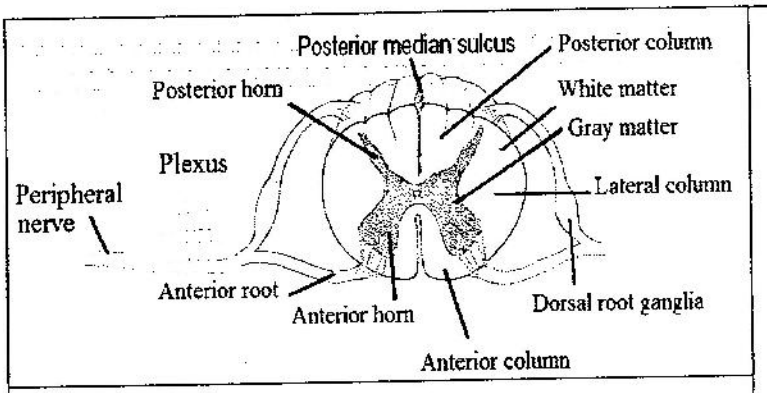
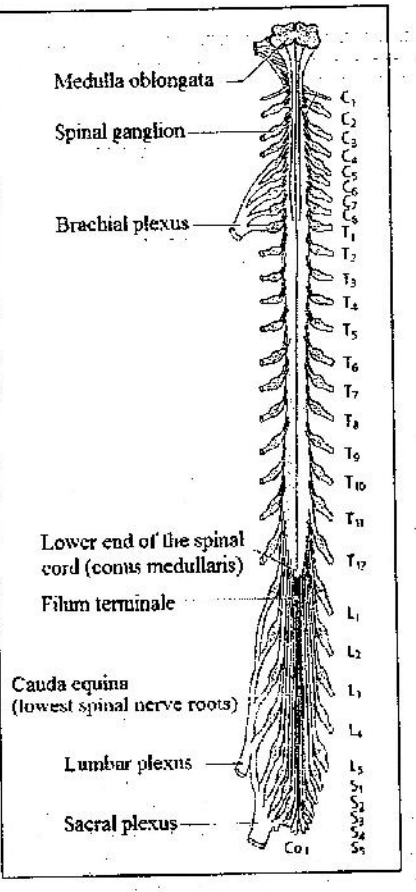


FIGURE 7 – 3. Anatomy of the spinal cord.



MEMBRANES OF THE BRAIN

Three membranes, the meninges, surround the brain to protect it from hard bones and blows to the head. The outermost is the densely fibrous and thick dura mater, which is closely applied to the inside of the bones and tightly adherent to the periosteum. Under the dura mater lies the middle layer, the thin and delicate arachnoid. The third and innermost layer is the very thin and sensitive pia mater, rich in capillaries, which is directly applied to the brain and dips into the fissures. Although the dura is very closely applied to the inside of the skull, it can become detached under certain conditions (hemorrhage), creating an epidural space (extradural space; e. g., epidural or extradural hemorrhage after skull fractures). The virtual space between the dura and the arachnoid (subdural space) is traversed by veins and can also be the site of clinically significant hemorrhage (subdural hemorrhage). The arachnoid and the pia mater are separated by a relatively wide space, the subarachnoid space, which is filled with cerebrospinal fluid (CSF). This clear fluid fills the whole subarachnoid space and so surrounds the brain like a protective cushion. When hemorrhage occurs in this space it is called subarachnoid hemorrhage. The pia mater is so closely adherent to the underlying brain that there is no space between them, the pia mater tightly holding the brain. The dura dips into the longitudinal cerebral fissure. This fold between the two hemispheres is known as the falx cerebri. The space between the cerebellum and the occipital lobe of the cerebrum over it is also lined with dura, which thus forms a tent-like cover over the cerebellum, the tentorium cerebelli. Finally, the dura dips between the cerebellar hemispheres to form the falx cerebelli. The membranes of the brain, the subarachnoid space, and the CSF pass through the foramen magnum in the base of the skull.

MEMBRANES OF THE SPINAL CORD

After passing through the foramen magnum, the membranes of the brain continue into the vertebral canal to become the membranes of the spinal cord, surrounding the spinal cord and the roots of the spinal nerves. The dura mater of the spinal cord forms a strong sac (dural sac) that is anchored around the foramen magnum and the intervertebral foramina and extends to the second sacral vertebra. In contrast to the dura of the brain, which is closely adherent to the periosteum of the skull, the dura of the spinal cord is not adherent to the bones of the vertebral canal, but separated from it by a space (epidural space, extradural space). This space is filled with fatty tissue and contains an extensive venous plexus. Inside the dura, the arachnoid is closely adherent to it, and is connected to the pia mater by delicate strands of connective tissue. The subarachnoid space, filled with CSF, lies between the arachnoid and the pia mater, which is closely applied to the surface of the spinal cord.

All spinal roots, including those in the cauda equina, are covered by the pia mater, which simply entrains the other two spinal membranes at the exit of the spinal nerves from the vertebral canal. Thus, the spinal nerves on leaving the vertebral canal are also covered by the dura and the arachnoid, which continue as the perineurium and epineurium.

CEREBROSPINAL FLUID (CSF)

The ventricular system consists of four cavities inside the brain, namely, the two lateral ventricles, the third ventricle, and the fourth ventricle. They are interconnected and filled with cerebrospinal fluid (CSF) which is a clear fluid, with a volume of about 150 ml that is produced by the choroid plexus in these cavities. From the lateral ventricles, the CSF flows through an opening in each side (interventricular foramen) and then through the foramen of Monro into the single median third ventricle. Eventually, the CSF produced in the ventricles flows through a narrow canal (cerebral aqueduct, aqueduct of Sylvius) into the single fourth ventricle, from which it flows through three openings in the roof of the fourth ventricle into the subarachnoid space and central canal of the spinal cord.

EXAMINATION OF THE NERVOUS SYSTEM

At first the neurologic techniques may seem difficult, but with practice, you will come to feel comfortable evaluating neurologic symptoms and disease. You should be active in your learning and ask your learner to review your skills.

HIGHER CEREBRAL FUNCTIONS

LEVEL OF CONSCIOUSNESS

Consciousness is a state of normal cerebral activity in which the patient is alert and aware of self and environment and is able to respond to internal changes e.g. hunger and to external changes. Level of consciousness primarily reflects the patient's capacity for arousal or wakefulness. It is determined by the level of stimuli needed for the patient to be aroused. The patients may fall into one of the following states:

1 Alert — When you start the examination and greet the patient in a normal voice, an alert patient opens eyes, looks at you, and responds fully and appropriately to stimuli.

2 Lethargic — When you start the examination and greet the patient in a normal voice, a lethargic patient is unresponsive. When you then greet the patient in a loud voice, a lethargic patient appears drowsy but opens eyes and looks at you, responds to questions, and then falls asleep.

3 Obtunded — Shake the patient gently as if awakening a sleeper. An obtunded patient opens the eyes and looks at you, but responds slowly and is somewhat confused. The patient immediately reverts to his or her prior level of consciousness when the stimulation ceases.

4 Stuporous — Only vigorous external stimuli can produce arousal e.g. squeezing the Achilles tendon, sternal pressure applied with a hard blunt object or supraorbital pressure from the candidate's thumb. These measures need of course permission from the examination committee.

5 Comatose — The patients do not open their eyes, utter sounds or obey commands in response to verbal or other stimuli.

Causes of altered consciousness are provided in BOX 7 – 1.

I SPOUT A VEIN

BOX 7 – 1. Causes of altered level of consciousness

Insulin: hypoglycemia or hyperglycemia	Opiate, drugs over dose and toxin	Epilepsy
Infection: encephalitis, meningitis	Uremia and other organ failure	Endocrine e.g. hypothyroidism
Stroke: hemorrhagic, thromboembolic	Trauma	Intracranial hemorrhage
Shock: hypovolemic, neurogenic, cardiogenic	Alcohol	Neoplasm
Psychosis	Vasculitis	

These terms are considered ill-defined terms nowadays and are replaced by the application of Glasgow coma scale (GCS). Glasgow coma scale (BOX 7 – 2) gives an indication of the patient's consciousness state and is not a substitute for neurological examination. It ranges from 3 – 15.

BOX 7 – 2. Glasgow coma scale

Eye-opening (E)		Best motor response (M)		Verbal response (V)	
Spontaneous	4	Obeys	6	Orientated	5
To speech	3	Localises	5	Confused conversation	4
To pain	2	Withdraws	4	Inappropriate words	3
Nill	1	Abnormal flexion (decorticate)	3	Incomprehensible sounds	2
		Extensor response (decerebrate)	2	Nil	1
		Nil	1		

GCS score = E + M + V	
Minimum	3
Maximum	15

SPEECH

If the patient is awake and alert, you can observe his/her speech for speech abnormalities in the form of dysphasia (difficulty in language), dysarthria (difficulty in articulation), or dysphonia (difficulty in phonation). Examination of speech should include searching for dysphasia, dysarthria, and dysphonia via the following two steps:

PART I: EXAMINATION OF LANGUAGE (FOR APHASIA (DYSPHASIA) AND PARAPHASIA)
In the vast majority of right handed people, the speech areas are in the left cortex. Two major cortical areas are involved in speech (FIGURE 7 – 4). These are:

1 The sensory speech area (Wernicke's area)

It is a portion of the parietal lobe which is responsible for reception, understanding and formulating coherent speech

2 The motor speech area (Broca's area)

It is located in the inferior portion of the frontal lobe which is responsible for initiation of complex series of movements necessary for speech

Aphasia (or dysphasia) is a disorder of the language content of speech. The prefixes dys- and a-, for aphasia or dysphasia are used interchangeably in clinical practice. Paraphasia is the supplementation of one word with another.
The main six parts of the language examination can be performed at the patient's bedside. These parts are:

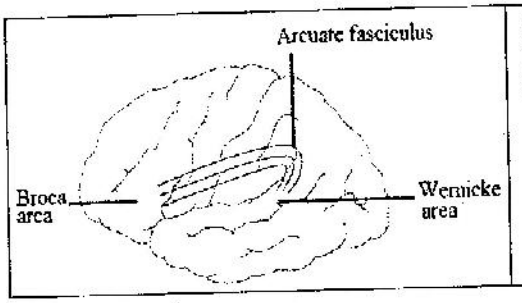


FIGURE 7 – 4. The speech areas.

1 COMPREHENSION

Give a simple three-stage command, e.g. pick up this piece of paper, fold it in half and place it under leg. Avoid visual clues by giving instruction from behind the patient's head.

2 FLUENCY

Ask the patient to tell you his/her story from the start

3 NAMING

Show the patient common objects, e.g. coin or pen, and ask its name. You need 20 objects. A deficit of naming (anomia) is the single most common finding in aphasic patients. When asked to name common objects (pencil or wristwatch), the patient may fail to come up with the appropriate word, may provide a circumlocutious description of the object ("the thing for writing"), or may come up with the wrong word (paraphasia). If the patient offers an incorrect but legitimate word ("pen" for "pencil"), the naming error is known as a semantic paraphasia; if the word approximates the correct answer but is phonetically inaccurate ("plentil" for "pencil"), it is known as a phonemic paraphasia

4 REPEATITION

Ask the patient to repeat a simple sentence, e.g. 'The boy went to school'

5 READING

Ask the patient to read motor acts that you had written e.g. raise your leg, raise your hand, or withdraw your legs. Alexia describes an inability to either read aloud or comprehend single words and simple sentences

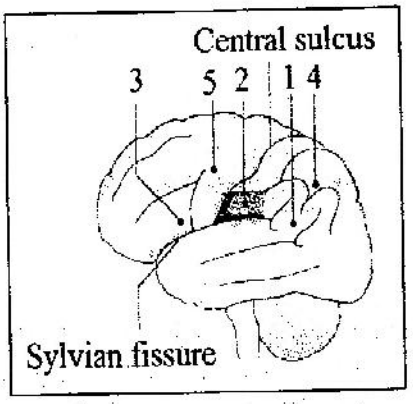
6 WRITING

Ask the patient to write his/her story from the start; examine his or her handwriting. Agraphia (or dysgraphia) is used to describe an acquired deficit in the spelling or grammar of written language

The candidate should then classify the patient's language by noting (the following FIGURE) that all have naming difficulty (anomia). Fluent aphasias arise from lesions posterior to the central fissure; repetition is affected by lesions around the Sylvian fissure.

1 Wernicke's aphasia: fluent aphasia with poor comprehension and poor repetition
2 Conduction aphasia: fluent aphasia with good comprehension and poor repetition
3 Broca aphasia: non-fluent aphasia with good comprehension and poor repetition
4 Transcortical sensory aphasia: fluent aphasia with poor comprehension and good repetition
5 Transcortical motor aphasia: non-fluent aphasia with good comprehension and good repetition

Large lesions affecting all of regions 1-5 cause global aphasia.



CLINICAL CLUE

Undergraduate students may face difficulty understanding all these aspects. They should understand the sequences of speech examination and at least three common types of dysarthria. Broca's dysphasia is also called motor or expressive dysphasia in which the patient understands you but can not speak. Wernicke's dysphasia is also called sensory or receptive dysphasia in which the patient can not understand you and talk a lot. Global aphasia is said to be present when the patient can not understand you and can not talk.

EXAMPLES

Examination of the language contents of speech had shown that the patient had good comprehension, fluency, naming, repetition, reading, and writing.

What is the interpretation?
Normal examination

Examination of the language contents of speech had shown that the patient had good comprehension, poor fluency, poor naming, poor repetition, poor reading, and good writing.

What Is the interpretation?
The interpretation is Broca's aphasia.

Examination of the language contents of speech had shown that the patient had poor comprehension, good fluency, poor naming, poor repetition, poor reading, and poor writing.

What is the interpretation?
The interpretation is Wernicke's aphasia.

Examination of the language contents of speech had shown that the patient had good comprehension, good fluency, poor naming, and poor repetition.

What is the interpretation?
The interpretation is conduction aphasia.

Examination of the language contents of speech had shown that the patient had poor comprehension, good fluency, poor naming, and good repetition.

What is the interpretation?
The interpretation is transcortical sensory aphasia.

Examination of the language contents of speech had shown that the patient had good comprehension, poor fluency, poor naming, and good repetition.

What is the interpretation?
The interpretation is transcortical motor aphasia.

PART II: EXAMINATION OF SPEECH (FOR DYSPHONIA (APHONIA) AND DYSARTHRIA)

Normal speech involves a highly coordinated sequence of contractions of the respiratory musculature, larynx, pharynx, tongue, and lips. These muscles are innervated by the phrenic, vagal, hypoglossal, and facial nerves. The nuclei of these nerves are controlled by the motor cortices through the corticobulbar tracts. Coordination of the movements of the articulatory structures requires simultaneous and coordinated activation of these cranial nerves, not only by direct cortical motor activation but also by indirect involvement of the basal ganglia and cerebellum. Thus, speech is impaired when any of the following structures are damaged or dysfunctional: motor cortex and its outflow tracts; cerebellum; basal ganglia; brain stem; peripheral portions of cranial nerves controlling the speech apparatus;

and target speech musculature, including facial muscles, pharynx, palate, tongue, vocal cords, diaphragm, and intercostal muscles. Examination sequence should include:

Listen to the patient's spontaneous speech, noting volume, rhythm, and clarity

Test function of different structures by asking the patient to repeat

1 Words with D, L and T (tongue function). Because our patients are Arabic, we commonly ask them to repeat the following sentence "La La La"
2 Words with P, B, or M (lip function). Because our patients are Arabic, we commonly ask them to repeat the following sentence "Me Me Me"
3 Al Mustanseria University (in Arabic) for scanning and monotonous speech
4 Ask the patient to count steadily to 30 to assess muscle fatigue
5 Ask the patient to cough

Both dysphonia (reduced ability to vocalize) and aphonia (inability to vocalize) are due to a disorder of the larynx or its innervation. Laryngitis is the most common cause, but these may result from other causes, such as damage to the superior laryngeal nerve, recurrent laryngeal nerve (vagal nerve palsy), or carcinoma involving the larynx. Dysphonic patients have a breathy sound resulting from abnormal apposition of the vocal cords. An aphonic patient is able to speak, but cannot produce any words or has a greatly diminished volume. Inability to abduct the vocal cords leads to a 'bovine' (and ineffective) cough. If the patient has a normal cough, this is hysterical. Dysarthrias on the other hand differ in the patterns of speech disturbance they produce and vary in their associated features.
Spastic dysarthria is also called upper motor neuron dysarthria, pseudobulbar palsy, supranuclear palsy, Donald duck speech, or hot potatoes speech and is caused by bilateral upper motor neuron disease or lesions of the corticospinal tract. In this condition, the tongue is small, the speech spastic and there is limited movement of the lips and mouth. Patients have particular difficulty with tongue function. The patient appears to be speaking from the back of the throat and it sounds as if the patient is trying to squeeze out words from tight lips. There is usually associated spasticity, a Babinski sign and increased jaw and pharyngeal reflexes. Flaccid dysarthria is also called lower motor neuron dysarthria, bulbar paralysis, or infranuclear speech. This is due to disease affecting motor neurons in the medulla and lower pons or their intramedullary or peripheral extensions. Speech becomes slurred and progressively less distinct. Lingual and labial consonants may become impossible with advancing paralysis. Speech has a nasal quality with bilateral paralysis of the palate. The voice may have a raspy quality because of vocal cord paralysis while facial muscle weakness causes slurred speech. Neurological examination may reveal atrophy and fasciculations of the tongue and weakness of the palate and the facial muscles. Extrapyramidal dysarthria produces hypophonic, monotonous, and low volume speech. Words run into one another and sentences start and stop suddenly. The ends of a sentence may be spoken explosively and palilalia is common (the constant repetition of a particular syllable). Ataxic (cerebellar) dysarthria is most commonly encountered with acute and chronic cerebellar disease. Cerebellar dysarthria may be slow and slurred similar to the speech of a drunken person. The difficulty in this instance is with the coordination of the muscles of speech and breathing. The patient's speech is irregular, slurred, changes from loud to soft, and is arrhythmic due to incoordination of speech with breathing. The words merge one with another or are spaced incorrectly. The speech may have a staccato or explosive quality with equal emphasis on all syllables (scanning). In Myasthenic dysarthria, the voice may be normal with rest or at the beginning of each sentence, but changes as speech progresses. Palatal weakness and hoarseness occur. When a history of variable dysarthria or dysphonia with prominent fatigability is elicited, a neuromuscular junction disorder such as myasthenia gravis should be considered. Myasthenia gravis is the most common cause of fatiguing speech

ORIENTATION

Orientation refers to the person's awareness of self in relation to other persons, places, and time. Orientation can be tested by directly questioning the patient about time, place and person:

1 TIME

Ask about the current day, month and year. If the patient is unable to know the month particularly in long standing illness where one day is much like the next, you can ask about the season (Autumn, Winter, Spring and Summer) and about the approximate time of day (morning, afternoon, evening or night).

2 PLACE

Every patient should know where he /she is by asking him (where are we now?)

3 PERSON

The patient should know name and address of his/her relatives. You can test this ability by directly asking the patient (who am I? who are you?) the answers likely are; my doctor, your patient or can mention his/ her name.

MEMORY

Memory can be defined as the ability of a person to retend the input received by the brain while something is scanned, evaluated and acted upon. Memory can be divided into at least three types; sensory, short term and long-term.

1 THE IMMEDIATE (SENSORY) MEMORY

It can be tested by asking the patient to repeat a name and address e.g. your name and address immediately. For example, tell the patient, (Repeat these words after me and remember them. I will ask for them later).

2 THE SHORT TERM MEMORY

If a given piece of data held in sensory memory is considered valuable enough, it is moved into short term memory in which information is limited by the number of bits of information that can be stored at any one time, which is approximately seven bits. Have you ever wondered why telephone numbers are seven digits? Five minutes after testing for the immediate memory, ask the patient, (What were those words I asked you to remember?)

3 LONG TERM MEMORY

Certain pieces of information are transferred from short term memory to long term memory. Rehearsal of information assists in such a transference. This type of memory can be tested by asking the patient about previous events happened before years e.g. Gulf war

CALCULATION ABILITY

This can be tested by asking the patient to subtract several sevens from 100 and at each step ask why. This test seems to be difficult even for normal individuals. Thus you ask the patient to subtract serial threes from 20.

ATTENTION AND CONCENTRATION

Attention can be defined as the ability of a person to focus on a matter. Concentration is the ability to sustain such a focusing. The previous task can provide valuable information about attention and concentration. Moreover the patient is asked to repeat a sequence of digits back words e.g. 195, the patient should say 591. Another useful fest is to ask the patient to spell a word back consisting of 5 digits back wards. In Arabic we use Halab = Balah.

REASONING AND JUDGMENT

Evaluation of judgment is performed by asking the patient to interpret a simple problem. Ask the patient the following:
What would you do if you see an addressed envelope with an unconcealed stamp on it on the street near a mailbox?
What would you do if you were in a crowded movie theater and a fire started?
A correct response to the first question would be to pick up the letter and mail it.
An example of an incorrect response might be, (I'd throw it in the trash).

PROVERB INTERPRETATION

Ask the patient what people mean when they use some of the following proverbs:

1 People who live in glass houses shouldn't throw stones
2 Don't count your chickens before they're hatched

Note the relevance of the answers and their degree of concreteness or abstractness. For example, "You should sew a rip before it gets bigger" is concrete, while "Prompt attention to a problem prevents trouble" is abstract. Average patients should give abstract or semiabstract responses. Concrete responses are often given by persons with mental retardation, delirium, or dementia, but may also be simply a function of limited education. Schizophrenics may respond concretely or with personal, bizarre interpretations.

CRANIAL NERVES EXAMINATION

Cranial nerves are twelve in number and originated from the brain stem in the following fashion:

1 Cranial nerves I and II are actually fiber tracts emerging from the brain
2 Medulla oblongata contains cranial nerves XII, XI, X, and IX
3 Pons contains cranial nerves VIII, VII, VI, and V
4 Midbrain contains cranial nerves IV and III

To remember the origin of the cranial nerves, they grouped from lower to upper level (from medulla oblongata to midbrain) like a football plan 4, 4, 2 (as seen above). The examination of the cranial nerves should be carried out in an orderly fashion.

CRANIAL NERVE I - OLFACTORY NERVE

The olfactory nerve supplies nerve endings to the superior nasal concha and upper one third of the nasal septum. In any patient with a suspected frontal lobe disorder, the olfactory nerve should be evaluated.
The examination sequence for olfaction (FIGURE 7 – 5):

1 STEP I Be sure that each nasal passage is open by compressing one side of the nose and asking the patient to sniff through the other or use a piece of cotton and ask the patient to blow from his/her nostril so as to move the cotton other wise, there is nasal obstruction
2 STEP II The patient should then close both eyes
3 STEP III Occlude one nostril and test smell in the other with familiar and nonirritating odors such substances as cloves, coffee, soap, or vanilla. The use of an irritating agent such as alcohol would involve cranial nerve V as well as cranial nerve I, and the test results would be inaccurate
4 STEP IV Ask each time if the patient smells anything and, if so, what
5 STEP V Test the other side

Anosmia refers to complete loss of sense of smell, hyposmia indicates diminished sense of smell, dysosmia is distorted sense of smell e.g. when pleasant odors smell unpleasant, and parosmia is a false smell, i.e. the subjective sensation of a smell which does not exist (i.e. hallucination). A unilateral loss of smell is more important than a bilateral loss because it indicates a lesion affecting the olfactory nerve or tract on that side. Anosmia or hyposmia has many causes including nasal disease, head trauma, smoking, aging, and the use of cocaine. It may be congenital. Dysosmia may be caused by purulent nasal infections or sinusitis. Transient parosmia may predate epileptic seizures of temporal lobe cortical origin (olfactory aura). This symptom may also be common amongst the normal population.

A B C

FIGURE 7 – 5. Examination of olfactory nerve with substance e.g. soap and tobacco (A) includes testing patency of each nostril (B) at first and then testing the sense of smell (C) of each nostril.

CRANIAL NERVE II - OPTIC NERVE

The optic nerve transmits the axons of the retinal ganglion cells. It begins at the back of the globe and passes through the optic canal of the sphenoid bone into the cranium where it joins with the contralateral nerve to form the optic chiasma. The optic tract then passes posteriorly to the lateral geniculate bodies of the thalamus then continues as optic radiation (FIGURE 7 – 9). The examination of optic nerve includes assessment of visual acuity, visual field, color vision, and ophthalmoscopic examination.

VISUAL ACUITY

Visual acuity is expressed as a ratio, such as 6/6. The number on the top is the distance between the patient and the chart that is usually 6 meters while the number below records the smallest line of letters read where each line of letters on the Snellen's chart is marked with a number 60, 36, 24, 18, 12, 9, and 6 and this represents the distance in meters at which a normally sighted person could read that line. Normal visual acuity is written as 6/6. The abbreviation OD refers to the right eye (oculus dexter); OS refers to the left eye (oculus sinister), and OU (oculus uterque) refers to both eyes.

I. USING STANDARD SNELLEN'S CHART

If a standard Snellen's chart is available, the patient should stand 6 meters from the chart. If the patient wears glasses, he or she should wear them for the examination. The patient is asked to cover one eye with the palm and read the smallest line possible. If Snellen's chart is not available, use a hand-held eye chart e.g. Rosenbaum card. The Rosenbaum card is a miniature, scale version of the Snellen chart for testing visual acuity at near. The card should be held in good light about 30 centimeters from the eye and record the vision in each eye.

II. EVALUATION OF PATIENTS WITH POOR VISION (FIGURE 7 – 6)

Patients with poor vision who are unable to read even the largest letter of the chart should be tested for finger counting ability. This crude measurement of visual acuity is obtained by holding up fingers in front of one of the patient's eyes with the other eye closed. The patient is then asked how many fingers are seen. If the patient is still unable to see, test for finger perception by covering one eye and directing the fingers in front of the other eye. If he/she is still unable to perceive fingers, it is important then to evaluate whether he or she has any light perception. This is performed by covering one eye and directing a light at the other eye. The candidate asks the patient whether he or she can see when the light is on and off. No light perception (NLP) is the term used when a person cannot perceive light. Impaired visual acuity can be due to lesions of the cornea, lens, vitreous, retina or optic nerve and the more distal visual pathway.

FIGURE 7 – 6. The clinical examination of visual acuity in patient with poor vision including finger counting (A), finger perception (B), and light perception (C).

VISUAL FIELDS

Assessment of the visual fields should include examination for sensory inattention, central visual field, examination of the blind spot, and peripheral visual field examination.

I. SENSORY INATTENTION (FIGURE 7 – 7)

Sit opposite the patient, about 1 arm length, eyes level and test for gross defects and visual neglect with both eyes open. Raise your arms up and out to the sides so that one hand is in the upper right quadrant of your vision and one in the upper left and ask the patient to look directly at you (look at my nose). Move one index finger and ask the patient, whilst looking straight at you, to point to the hand which is moving. Test with the right, left, and then both hands and then test the lower quadrants in the same way. Note whether the patient reports seeing only one side move and which quadrant or side is affected. If visual neglect due to non-dominant parietal lobe lesion is present, the patient will be able to see each hand moving individually but reports seeing only one hand when both are moving.

II. CENTRAL VISUAL FIELD (FIGURE 7 – 7)

The normal central vision extends about 30° in all directions of central fixation. Use a red hatpin to test one eye qualitatively at a time and ask the patient to cover one eye and look directly at you. Shut your eye that is opposite the patient's covered eye and hold the hatpin in the centre of the visual field, as close to fixation as possible. Ask the patient what color the hatpin is. If it is pale or pink, this implies color esaturation, usually affecting the optic nerve. Compare the four quadrants of the visual field centrally; each time ask about color esaturation. Note that the visual field for red may be smaller than for white. Central visual field restriction will result in central scatoma.

III. BLIND SPOT

Within the normal field of each eye is a 5-degree blind spot that is 15 – 20 degrees temporal to central fixation, corresponding to the optic disc, which lacks receptor cells. This is a physiological scotoma corresponding to absence of photoreceptors where the optic nerve leaves the eye. Ask the patient to cover one eye and look directly at you and shut the eye that is opposite the patient's covered eye. Hold the hatpin at the fixation point; you and the patient focus on each other's eye. Move the hatpin temporally and horizontally until it disappears from your visual field. Maintaining the same temporal horizontal position, move it anteriorly or posteriorly until it also disappears from the patient's visual field (FIGURE 7 – 7). Compare the size of the patient's blind spot to yours.

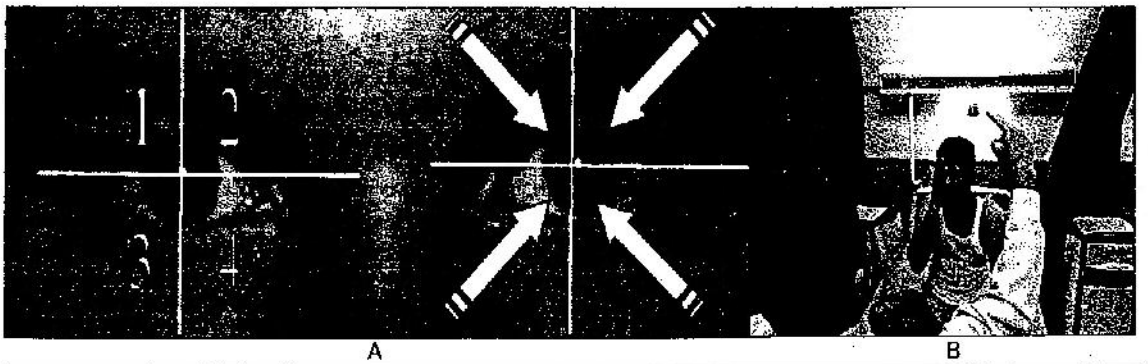
A B C

FIGURE 7 – 7. The clinical examination of visual fields sensory inattention (A). The patient should open both eyes and look at you while you asking him about which of the finger is moved. Central visual field (B) is usually restricted to 30 degrees from the central fixation in all directions. The blind spot examination (C) includes asking the patient to look at you while you move the red hatpin temporally away from the central fixation and asking the patient about the point of disappearance.

IV. EXAMINATION OF THE PERIPHERAL VISUAL FIELD BY CONFRONTATION TEST (FIGURE 7 – 8)

The normal monocular visual field subtends an angle of about 160° in the horizontal plane and about 135° in the vertical plane. With binocular vision, the horizontal range of vision exceeds 180°.

The candidate sits at about arm length from the patient, with the eyes of both patient and candidate aligned in the horizontal plane. The eye not being tested is covered by the patient's hand or an eye patch. The candidate closes the eye opposite the patient's covered eye, and the patient is instructed to fix on the candidate's open eye. Now the monocular fields of the patient and the candidate are superimposed, which allow comparison of the patient's field with the candidate's presumably normal field. Examine the outer aspects of the visual field with a waggling index finger or a fine probe with a large red or white head pin prick. Bring the test object into the field of vision approach from the periphery at several points on the circumference i.e. superior nasal quadrant, superior temporal quadrant, inferior nasal quadrant, and inferior temporal quadrant of the visual fields. Ask the patient to respond as soon as the movement of the test object is observed. The procedure is then repeated for the patient's other eye.



A B

FIGURE 7 – 8. The clinical examination of peripheral visual fields. The first view (A) illustrates the quadrants of the peripheral visual field that are:

(1) The upper temporal quadrant.
(2) The upper nasal quadrant.
(3) The lower temporal quadrant.
(4) The lower nasal quadrant.

The arrows pointing to the directions of waggling finger or pin head.
The second view (B) illustrates the technique of examination.

Hemianopia is a field defect that encompasses roughly half of the field. Vertical hemianopia can be nasal or temporal. When only one-fourth of the field is affected, the resulting deficit is called quadrantanopia.
Bilateral field defects are said to be homonymous when they are similarly located in both visual fields (the right or left side of vision in both eyes is lost e.g. the nasal field in the right eye and the temporal field in the left eye) while if the visual field is affected on different part in each eye, the term is heteronymous (bitemporal or binasal). They are congruous when there is a point-to-point correspondence of the defect in either field; otherwise they are called incongruous. The exact pattern of peripheral visual field loss may have localizing value due to the retinotopic arrangement of fibres in the visual pathways: any unilateral area of restricted loss implies a prechiasmatic lesion (choroid, retina, optic nerve). Bilateral homonymous scotomata are postchiasmal in origin; bilateral heteronymous scotomata may be seen with chiasmal lesions. Optic tract gives rise to homonymous hemianopia that is usually incongruous. Optic radiation lesion or occipital cortex defect gives rise to homonymous hemianopia that is usually congruous. The most common visual field abnormalities are illustrated in FIGURE 7 – 9.

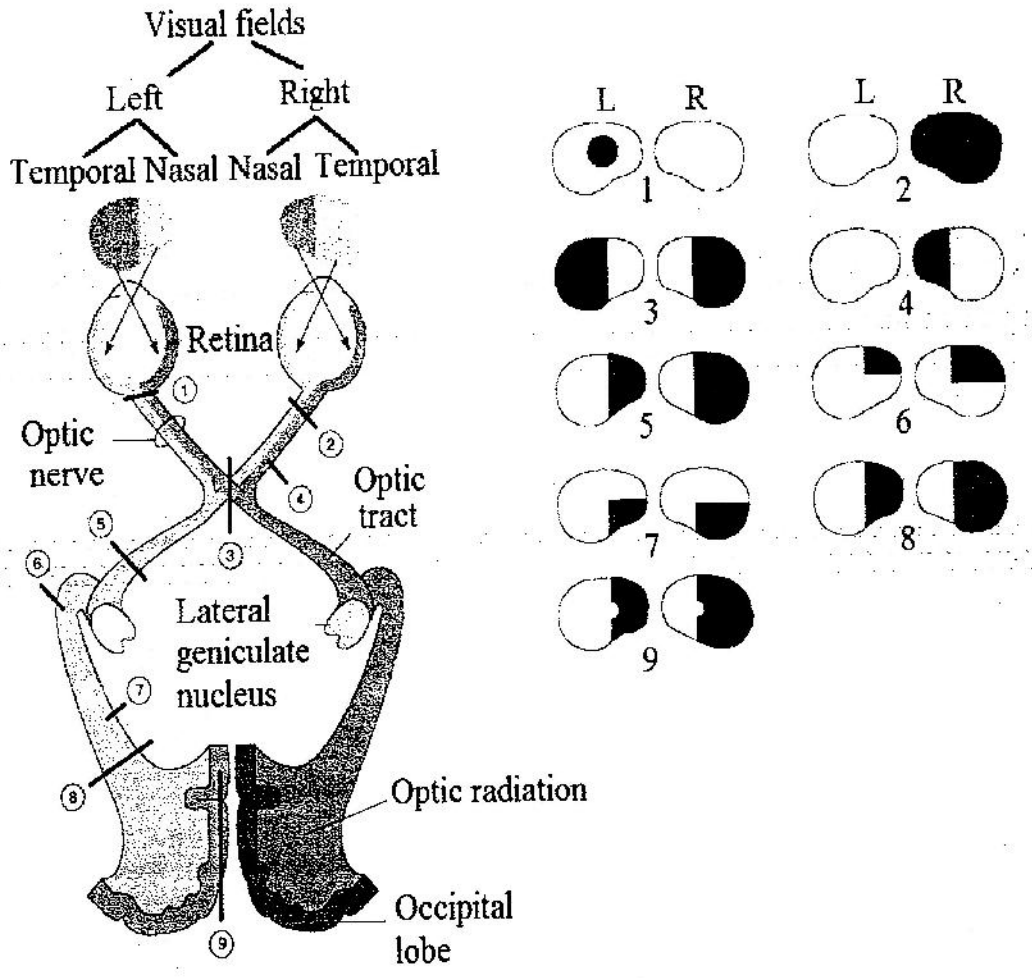


FIGURE 7 – 9. Common visual field abnormalities:
1. Central scotoma
2. Total blindness of the right eye from a complete lesion of the right optic nerve.
3. Bitemporal hemianopia caused by pressure exerted on the optic chiasm by a pituitary tumor.
4. Right nasal hemianopia caused by a perichiasmal lesion (e.g., calcified internal carotid artery).
5. Right homonymous hemianopia from a lesion of the left optic tract.
6. Right homonymous superior quadrantanopia caused by partial involvement of the optic radiation by a lesion in the left temporal lobe (Meyer loop).
7. Right homonymous inferior quadrantanopia caused by partial involvement of the optic radiation by a lesion in the left parietal lobe.
8. Right homonymous hemianopia from a complete lesion of the left optic radiation. (A similar defect may also result from lesion 9.)
9. Right homonymous hemianopia (with macular sparing) resulting from posterior cerebral artery occlusion.

CLINICAL CLUE

Homonymous hemianopia
Incongruent defects: lesion of the optic tract
Congruous defects: lesion of the optic radiation or occipital cortex
Macula sparing: lesion of the occipital cortex

Homonymous quadrantanopia
Superior: lesion of the temporal lobe
Inferior: lesion of the parietal lobe
Mnemonic is PITS (parietal – inferior, temporal – superior).

Bitemporal hemianopia
Lesion of the optic chiasm
Upper quadrant defect > lower quadrant defect = inferior chiasmal compression, commonly pituitary tumor
Lower quadrant defect > upper quadrant defect = superior chiasmal compression, commonly craniopharyngioma

Central scotomata, or loss of central (macular) vision, may be due to demyelination of the optic nerve (multiple sclerosis causes unilateral or asymmetrical bilateral scotomata); toxic causes, such as methyl alcohol (symmetrical bilateral scotomata); nutritional causes, such as tobacco or alcohol (symmetrical central scotomata); vascular lesions (unilateral); and gliomas of the optic nerve (unilateral).

CLINICAL CLUE

Concentric diminution of the field (tunnel vision) may be caused by glaucoma; retinal abnormalities such as chorioretinitis or retinitis pigmentosa; papilloedema; or acute ischemia, as with migraine.

Foster-Kennedy Syndrome consists of unilateral optic atrophy, contralateral papilloedema, central scotoma, anosmia (variable), and systemic symptoms such as headache, dizziness, vertigo, and vomiting. It is caused by meningioma of optic nerve, olfactory groove or sphenoid wing and frontal lobe tumour.

COLOR VISION

This test assesses the function of the retinal cones and optic nerve using Ishihara plates. This test is not a routine examination. Abnormality of the color vision may indicate congenital red/green color blindness, age related macular disease, and optic nerve disease.

OPHTHALMOSCOPIC EXAMINATION

This examination is mainly designed for postgraduate students who may consult a new book, Rajooj's clinical ophthalmoscopic examination. Undergraduate students should follow the instructions provided in their medical schools about the need of ophthalmoscopic examination.

CRANIAL NERVES III, IV, AND VI - OCULOMOTOR, TROCHLEAR, AND ABDUCENS

The oculomotor nerve supplies the medial, superior, and inferior rectus muscles and the inferior oblique muscle, which control most eye movements. The third nerve also innervates the intrinsic muscles, controlling pupillary constriction and accommodation. The trochlear nerve is responsible for movement of the superior oblique muscle. The abducens nerve is responsible for movement of the lateral rectus muscle.
These cranial nerves are usually examined togather. The sequence of examination (Remember 3 X 3):

STEP I	STEP II	STEP III
Prime position	Ocular movements (H-test)	Pupillary reaction
Ptosis	Nystagmus	Direct test
Proptosis	Eye limitation	Indirect test
Pupillary inequality (anisocoria)	Diplopia	Near (accommodation) reflex

I. PRIME POSITION (FIGURE 7 – 10)

Place a finger or a pen, about arm length, in front of the patient's head and look for ptosis, proptosis, and anisocoria. Ptosis is a dropping of the upper eye lid (BOX 7 – 3) which can be partial or complete. Proptosis means a bulging of the eye ball that occurs in thyroid eye disease, orbital cellulitis and hematoma, Wegener's granulomatosis, and in orbital meningioma.

FIGURE 7 – 10. Examination of prime position.

The normal pupil is round, regular, and centered within the iris; its size varies with age and with the intensity of ambient light. In a brightly illuminated examining department, normal pupils are about 3 mm in diameter in adults. They are often smaller in the elderly and commonly 5 mm or more in diameter in children. Pupillary size may be asymmetric in 20% of the population (physiologic anisocoria), but the difference in size is not more than 1 mm.

BOX 7 – 3. Causes of ptosis

Bilateral ptosis		Unilateral ptosis
Myotonia dystrophica	Syphilis	As for bilateral ptosis
Myasthenia gravis	Congenital	Third cranial nerve palsy
		Horner's syndrome

Anisocoria means inequality of the pupil. It may occur normally in 20% of population or may occur due to drugs, blunt trauma to the eye, glaucoma, oculomotor nerve paralysis and Horner's syndrome.

Horner's syndrome comprises miosis (small pupil), ptosis, enophthalmos (sunken eye), and anhydrosis (loss of sweating) on one side. To distinguish the causes, look at:

1. Heterochromia (difference in iris colour) is seen in congenital Horner's.
2. Anhydrosis as in BOX 7 – 4.

BOX 7 – 4. Causes of Horner's syndrome

Central lesions	Pre-ganglionic lesions	Post-ganglionic lesions
Anhydrosis of the face, arm and trunk	Anhydrosis of the face	No anhydrosis
Stroke Syringomyelia Multiple sclerosis	Pancoast's tumour Thyroidectomy Trauma	Carotid artery aneurysm or dissection Cavernous sinus thrombosis Cluster headache

The neurotransmitter agent that causes pupillary Dilation is aDrenaline and that causes pupillary Constriction is acetylCholine

Pupillary enlargement, or mydriasis, is associated with use of sympathomimetic agents, anticholinergics e.g. atropine or tropacamide, third cranial nerve palsy, Holmes Adie pupil, midbrain lesion, pheochromocytoma, and with acute angle closure glaucoma. Pupillary constriction, or miosis, occurs with the use of parasympathomimetics, opiate, organophosphorus drugs, Horner's syndrome, Argyll Robertson pupil, and pontine lesion.

II. OCULAR MOVEMENTS (H – test)

Ocular movements are effected by the contraction and relaxation of the extraocular muscles. This results in simultaneous movement of the eyes up or down or from side to side as well as in convergence. A useful mnemonic to remember the cranial nerves supply for extra-ocular muscles is (SO4 L6 / 3) where superior oblique muscle is supplied by the fourth cranial nerve; lateral rectus is supplied by the sixth cranial nerve; the remaining muscles are supplied by the third cranial nerve. An important cause of a deviated eye is paretic (weak), or paralyzed, extra-ocular muscle(s). Paralysis of these muscles is detected by H – test examination of the six diagnostic positions of gaze. The examination sequences are as follow:

FIGURE 7 – 11. The technique for H – test examination.

1 Hold the patient's chin steady (FIGURE 7 – 11) with the left hand, and ask the patient not to move his/her head and to follow the right hand.
2 Hold the right index finger about arm length from the patient's nose. From the midline, the patient is asked to follow the finger moving to the right and up and down (FIGURE 7 – 12).
3 Then reverse your hands, now holding the chin with your right hand. Cross the midline and repeat the finger movement on the other side.
4 Observe the movement of both eyes, which should follow the finger smoothly. Look for nystagmus and eye limitation in all movements and ask the patient about diplopia.

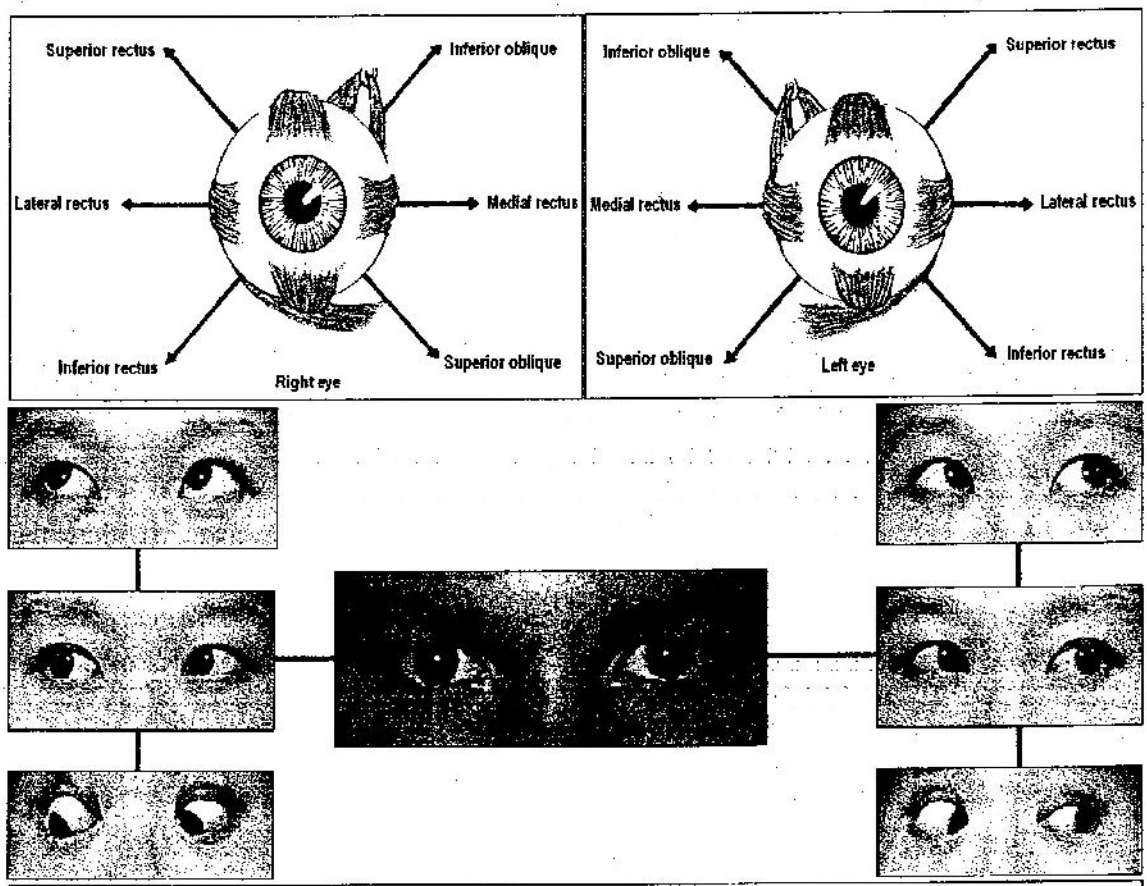


FIGURE 7 – 12. H – test technique showing the positions of the eyes during the test and the corresponding extra-ocular muscles.

Occasionally when looking to the extreme side, the eyes develop a rhythmic motion called end-point nystagmus.

THIRD CRANIAL NERVE PALSY

A complete lesion of the oculomotor nerve produces closure of the affected eye because of impaired levator function. Passively elevating the paralyzed lid (FIGURE 7 – 13) shows the involved eye to be laterally deviated because of the unopposed action of the lateral rectus muscle, which is not innervated by the oculomotor nerve. On attempted downgaze, the unaffected superior oblique muscle, which is innervated by the trochlear (IV) nerve, causes the eye to turn inward. Diplopia is present in all directions of gaze except for lateral gaze toward the side of involvement. Third nerve lesions are generally divided into medical or surgical causes. The pupillary involvement and the degree of ptosis are helpful in differentiating the two. Medical third nerve lesions spare the pupil and have a greater degree of ptosis (infarction of the nerve). The combination of headache and third nerve palsy (painful third cranial palsy) should raise the possibility of posterior communicating artery aneurysm (BOX 7 – 5).

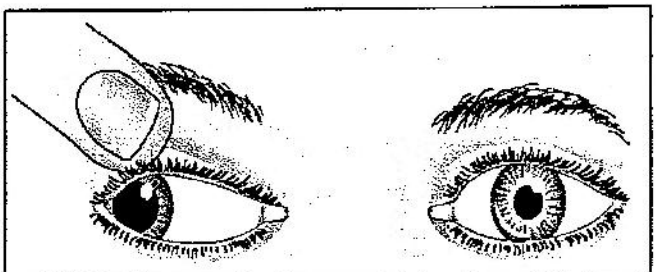


FIGURE 7 – 13. The clinical features of third cranial nerve palsy.

BOX 7 – 5. Causes of third cranial nerve palsy

Diabetes mellitus	Cavernous sinus thrombosis
Vasculitis e.g. temporal arteritis, SLE	Weber's syndrome
Posterior communicating artery aneurysm (pupil dilated)	Multiple sclerosis

FOURTH CRANIAL NERVE PALSY

The lesion results in paralysis of the superior oblique with weakness of downward (and outward) movement. The patient may walk around with his or her head tilted away from the lesion-that is, to the opposite shoulder (this allows the patient to maintain binocular vision). Diplopia is most pronounced when the patient looks downward with the affected eye adducted (as in looking at the end of one's nose). The clinical features are shown in FIGURE 7- 14.

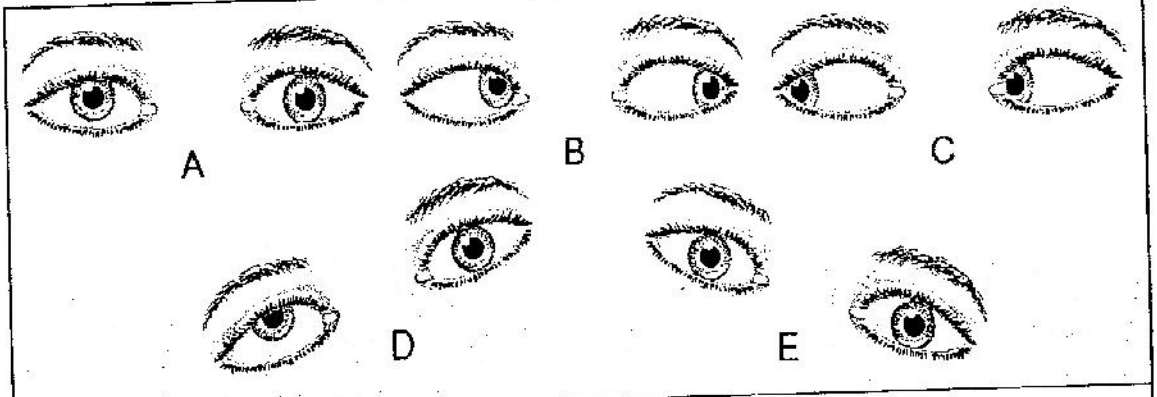


FIGURE 7 – 14. Clinical findings with trochlear (IV) nerve lesion include that the affected (right) eye is elevated on primary forward gaze (A). The extent of elevation is increased with adduction (B) and decreased with abduction (C). Elevation is greatest when the head is tilted toward the side of the involved eye (D) and decreases with head tilting in the opposite direction (E).

BIELSCHOWSKY'S SIGN, BIELSCHOWSKY'S TEST

Bielschowsky's sign is head tilt towards the opposite shoulder, typically contralateral to a trochlear (IV) nerve palsy. Bielschowsky's (head tilt) test consists of the candidate tipping the patient's head from shoulder to shoulder to see if this improves or exacerbates double vision, as will be the case when the head is, respectively, tilted away from or towards the affected side in a unilateral trochlear (IV) nerve lesion.

SIXTH NERVE LESION

There are failure of lateral movement, convergent strabismus and diplopia. These signs are maximal on looking to the affected side (FIGURE 7 – 15).

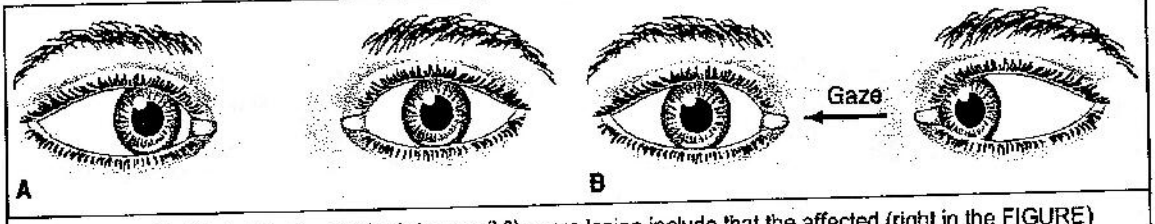


FIGURE 7 – 15. Clinical findings with abducens (VI) nerve lesion include that the affected (right in the FIGURE) eye is adducted at rest (A) and cannot be abducted (B).

The eyes are normally maintained at rest in the middle line by the balance of tone between opposing ocular muscles. Disturbance of this tone, which depends on impulses from the retina, the muscles of the eyes themselves and various vestibular and central connections, allows the eyes to drift in one direction. This drift is corrected by a quick movement (saccadic) back to the original position. When these movements occur repeatedly nystagmus is said to be present. The direction of the nystagmus is defined as that of the fast (correcting) movement, although it is the slow drift that is abnormal. Nystagmus from any cause tends to be accentuated by gaze in a direction away from the middle line. Nystagmus is classified into jerky and pendular nystagmus. Jerky horizontal nystagmus may be due to (i) a vestibular lesion (acute lesions cause nystagmus away from the side of the lesion white chronic lesions cause nystagmus to the side of the lesion); (ii) a cerebellar lesion (unilateral diseases cause nystagmus to the side of the lesion); (iii) toxic causes, such as phenytoin and alcohol (may also cause vertical nystagmus but less often); and (iv) internuclear

ophthalmoplegia. Jerky vertical nystagmus (Vertical nystagmus means nystagmus where the oscillations are in a vertical direction) may be due to a brainstem lesion. Upbeat nystagmus suggests a lesion in the midbrain or floor of the fourth ventricle, while downbeat nystagmus suggests a foramen magnum lesion (Arnold – Chiari malformation). Phenytoin or alcohol can also cause this abnormality.

With pendular nystagmus the nystagmus phases are equal in duration. Its cause may be retinal (decreased macular vision, e.g. albinism) or congenital. This condition is thought to occur as a result of poor vision or increased sensitivity to light. It develops in childhood and occurs as the patient performs searching movements in an attempt to fixate or improve the visual impulses. Nystagmus confined to one eye (unilateral nystagmus) suggests a peripheral lesion of the nerve or the muscle or a lesion of the medial longitudinal bundle.

Ophthalmoparesis is a weakness or limitation, ophthalmoplegia a paralysis, of eye movements. Two types are clinically relevant. These are:

1. External ophthalmoplegia which means weakness of the extra-ocular muscles of central, neuromuscular, or myopathic origin. The causes are:
   - Supranuclear: e.g. progressive supranuclear palsy.
   - Nuclear: e.g. internuclear ophthalmoplegia (INO).
   - Cranial nerve palsy: III, IV, VI, or combinations of them.
   - Neuromuscular junction: myasthenia gravis.
   - Extraocular muscles: e.g. thyroid ophthalmopathy.
2. Internal ophthalmoplegia which means fixity of the pupil with loss of all pupillary reflexes (iridoplegia) and ciliary apparatus. Hence in an oculomotor (III) nerve palsy there may be both internal and external ophthalmoplegia.

III. PUPILLARY REACTION

This part of examination includes evaluation of light reflex (direct and indirect) and near (accommodation) reflex. The afferent limb of the pupillary reflex involves the optic nerve and the efferent limb of the reflex arc involves the third cranial nerve.

PUPILLARY LIGHT REFLEX

The candidate should ask the patient to look in the distance while he/she shines a bright light in the patient's eye (FIGURE 7 – 16). The light source should come from the side, the nose being used as a barrier to light to the other eye. The candidate should observe the direct and consensual (indirect) pupillary responses. The candidate then repeats the test on the other eye. The normal response is pupillary constriction of the eye subjected to light (direct) and pupillary constriction of the other eye (indirect).

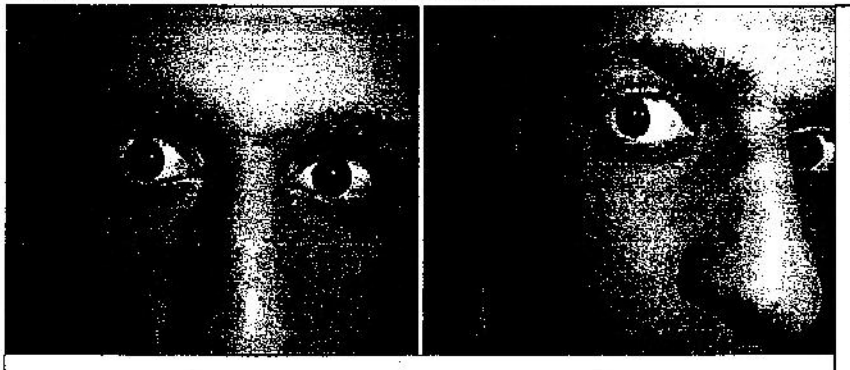


FIGURE 7 – 16. The pupils are normally dilated in the dark (A) and constrict in response to light (B).

SWINGING LIGHT REFLEX

The swinging light test is a modification for testing the pupillary light reflex. This test reveals differences in the response to afferent stimuli of the two eyes. The patient fixates on a distant target while the examiner rapidly swings a light from one eye to the other, observing for constriction of the pupils. Normally there is pupil constriction which is greater with the direct light test than the indirect. In some conditions, there is a paradoxical dilatation of the pupil on which the light is shone. This condition, called a Marcus Gunn pupil or afferent pupillary defect, is associated with an afferent limb defect in the eye being illuminated i.e. optic nerve problem. Marcus Gunn pupil is of two types; relative and complete. In relative afferent pupillary defect, one pupil constricts less markedly in response to direct illumination than to illumination of the contralateral pupil. Relative afferent pupillary defect is commonly associated with disorders of the ipsilateral optic nerve, which interrupt the afferent limb and affect the pupillary light reflex. In complete afferent pupillary defect, when an eye has optic atrophy (blind eye), the affected pupil will dilate paradoxically after a short time when the torch is moved from the normal eye to the abnormal eye. It occurs because the affected eye had no afferent reflex (optic nerve damage) but stills receive efferent reflex from the third cranial nerve. So that when the light is shone to the normal eye, there is normal response in both pupils which is constriction but when the light is now shone to the abnormal eye there will be pupillary dilation because the afferent limb is damaged and the indirect light reflex time is finished.

NEAR (ACCOMMODATION) REFLEX (FIGURE 7 – 17)
The near reflex is tested by having the patient look first at some distant target and then at a target placed about 5 inches away from his or her nose. When the patient focuses on the near target, the eyes should converge, the pupils should constrict, and there is a ptosis of the upper eyelids.

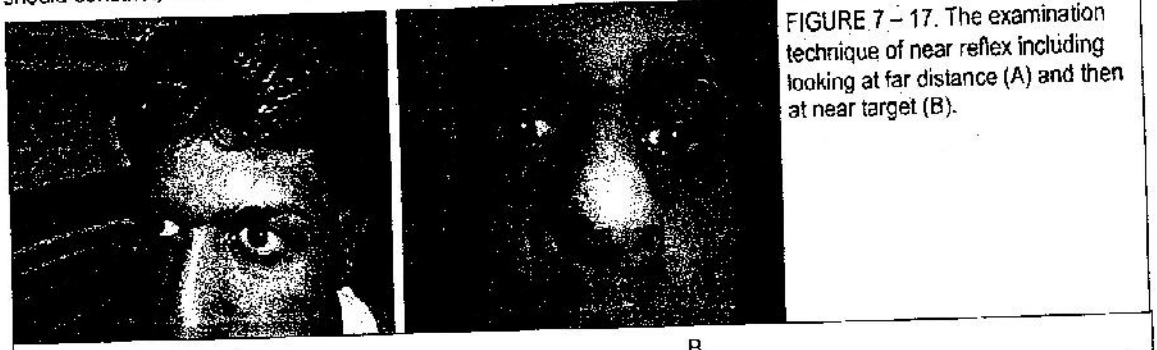


FIGURE 7 – 17. The examination technique of near reflex including looking at far distance (A) and then at near target (B).

Pupillary abnormalities are often markers of neurologic disease. A condition known as Holmes Adie's pupil is a pupil dilated 3–6 mm that constricts little in response to light and accommodation. This pupil is often associated with diminished to absent deep tendon reflexes in the extremities (FIGURE 7 – 18). Argyll Robertson pupil is a pupil constricted 1–2 mm that reacts to accommodation but is nonreactive to light. It is associated with neurosyphilis. Horner's syndrome is sympathetic paralysis of the eye that is due to interruption of the cervical sympathetic chain. Diabetic mononeuropathy is the most common cause for oculomotor nerve, trochlear nerve, or abducens nerve palsy.

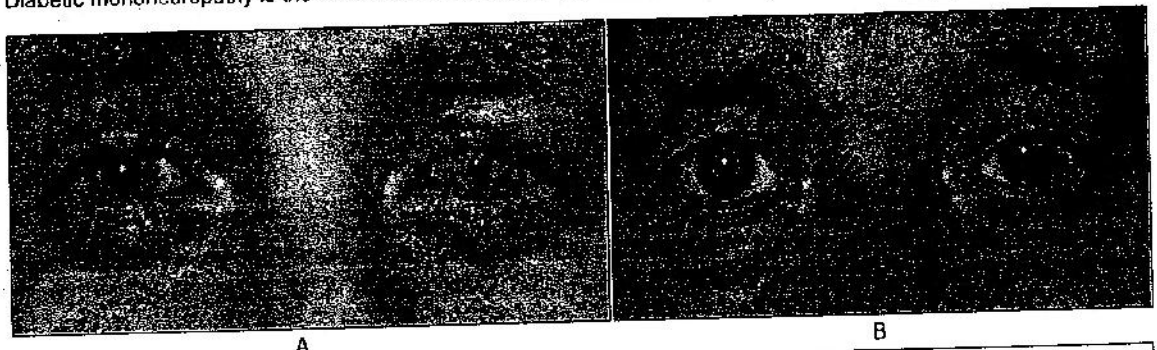


FIGURE 7 – 18. Pupillary abnormalities including Holmes Adie pupil (A) and Horner's syndrome (B).

Internuclear ophthalmoplegia (INO) (FIGURE 7 – 19) results from damage to the medial longitudinal fasciculus that ascends from the abducens nucleus in the pons to the oculomotor nucleus in the midbrain. The resulting clinical picture includes failure of ipsilateral adduction on attempted lateral gaze and nystagmus in the abducted eye. Multiple sclerosis is the most common cause, but tumor, stroke, trauma, or any brainstem process may be responsible.

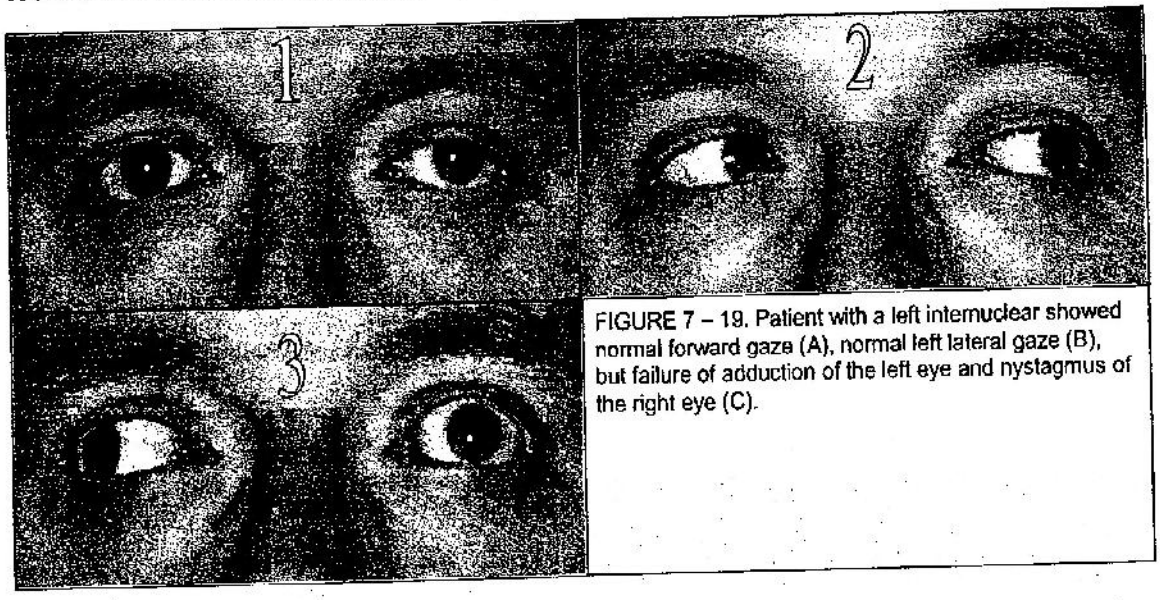


FIGURE 7 – 19. Patient with a left internuclear showed normal forward gaze (A), normal left lateral gaze (B), but failure of adduction of the left eye and nystagmus of the right eye (C).

Patient with bilateral injury to the medial longitudinal fasciculus will have bilateral internuclear ophthalmoplegia. One and a half syndrome is due to a combined lesion of the medial longitudinal fasciculus and the abducens nucleus on the same side. One-and-a-half syndrome (FIGURE 7 – 20) is rare but important to recognise. These patients have horizontal gaze palsy when looking to one side (the 'one') plus impaired adduction on looking to the other side (the 'and-a-half'). Therefore the patient's only horizontal eye movement is abduction of the eye on the other side. Vertical movement is unaffected. One-and-a-half syndrome can be caused by a stroke (infarct), plaque of multiple sclerosis or tumour in the dorsal pons.

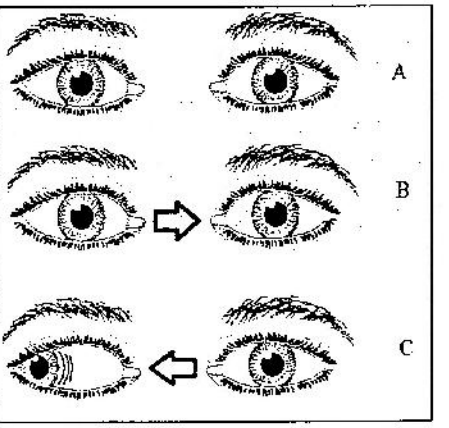


FIGURE 7 – 20. Patient with one and a half syndrome showed normal forward gaze (A), failure of all horizontal eye movement (B), and failure of adduction of the ipsilateral eye (C).

LIGHT NEAR DISSOCIATION

Impaired pupillary reactivity to light, with preserved constriction during accommodation (light near dissociation) is usually bilateral and may result from (BOX 7 – 6) neurosyphylis, diabetes, optic atrophy, and tumors compressing the midbrain tectum. Diphtheria causes preserved light reflex but impaired accommodation reflex.

BOX 7 – 6. Light near dissociation

Light	Accommodation	Causes
-	+	Diabetes mellitus Neurosyphylis (Argyll-Robertson pupil) Tumors compressing midbrain tectum
+	-	Diphtheria

EXAMINATION OF DIPLOPIA

Diplopia means double vision. Most cases of diplopia (about 60%) are not due to a cranial nerve abnormality. First find out whether the diplopia is monocular or binocular. Monocular diplopia persists when one eye is covered. It is usually due to an eye problem such as astigmatism, dislocated lens, uneven contact lens surface or thick spectacles. If the diplopia is binocular, consider the common causes:

1. Supranuclear: e.g. progressive supranuclear palsy.
2. Nuclear: e.g. internuclear ophthalmoplegia (INO).
3. Cranial nerve palsy: III, IV, VI, or combinations of them.
4. Neuromuscular junction: myasthenia gravis.
5. Extraocular muscles: e.g. thyroid ophthalmopathy.

Diplopia can be an early sign of ocular muscle weakness because the light falls on different parts of the corresponding retinas due to slight movement differences. If diplopia is present, further testing is necessary. The false image is usually paler, less distinct and always more peripheral than the real one. Ask the patient whether the two images lie side by side or one above the other. If they are side by side, only the lateral or medial recti can be responsible. If they lie one above the other, then either of the obliques or the superior or inferior recti may be involved. To decide which pair of muscles is responsible, perform H – test and ask the patient in which direction there is maximum image separation. Separation is greatest in the direction in which the weak muscle has its purest action. At the point of maximum separation, cover one eye and find out which image disappears. Loss of the lateral image indicates that the covered eye is responsible.

CLINICAL CLUE

Sometimes the candidate is asked to individually examine third, fourth, and sixth cranial nerves:

1. Third cranial nerve is examined in same way of prime position, H – test, and pupillary reaction.
2. Fourth cranial nerve is examined via prime position, H – test (or by asking the patient to turn the eye in and then to try to look down) and by performing Bielschowsky's test.
3. Sixth cranial nerve is examined via prime position and by performing H – test (or asking the patient to turn the eye horizontally out).

CRANIAL NERVE V - TRIGEMINAL NERVE

The trigeminal nerve is responsible for supplying sensation to the face, the nasal and buccal mucosa, and the teeth. The motor division supplies the temporalis, masseter, medial and lateral pterygoids (muscles of mastication), and some smaller muscles not examined clinically. The three major subdivisions of the trigeminal nerve are the ophthalmic, the maxillary, and the mandibular (FIGURE 7 – 21).

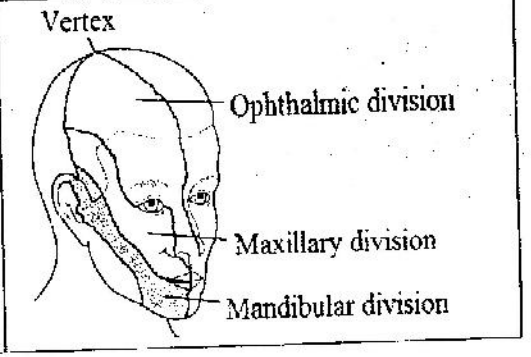


FIGURE 7 – 21. Divisions of the trigeminal nerve.

The examination of the trigeminal nerve consists of the following:

1 Testing the sensory function
2 Testing the motor function
3 Testing the reflexes

TEST SENSORY FUNCTION (FIGURE 7 – 22)
The sensory function of the trigeminal nerve is tested by asking the patient to close the eyes and to respond when a touch is felt. A wisp of cotton wool is applied on all three subdivisions of the nerve of one side and then to the corresponding position on the other side. The patient is also asked if one side feels the same as or different from the other side. The procedure is repeated using a sharp pin, alternating between sides.

FIGURE 7 – 22. Examination of the sensory function of the trigeminal nerve including light touch (A) and pin prick sensation (B).

CLINICAL CLUE

Remember that if there is total loss of sensation in all three divisions of the nerve, this suggests that the level of the lesion is at the ganglion or the sensory root e.g. acoustic neuroma. If there is total sensory loss in one division only, this suggests a postganglionic lesion. The ophthalmic division is most commonly affected because it runs in the cavernous sinus and through the orbital fissure, where it is vulnerable to a number of different insults.

TEST MOTOR FUNCTION (FIGURE 7 – 23)
The examination should be performed in the following steps:

1 Inspect muscles of mastication in order to look for wasting that is easily seen in the temporalis muscles above the zygomatic arch as well as the masseter muscles
2 Ask the patient then to clench the teeth and palpate for contraction of the masseter above the mandible
3 Ask the patient to open his/her mouth to see any deviation in the jaw movement. A unilateral lesion of the motor division causes the jaw to deviate towards the weak (affected) side
4 Ask the patient now to open his mouth against resistance i.e. against your hand to detect any deviation. A unilateral lesion of the motor division causes the jaw to deviate towards the weak (affected) side

A B C

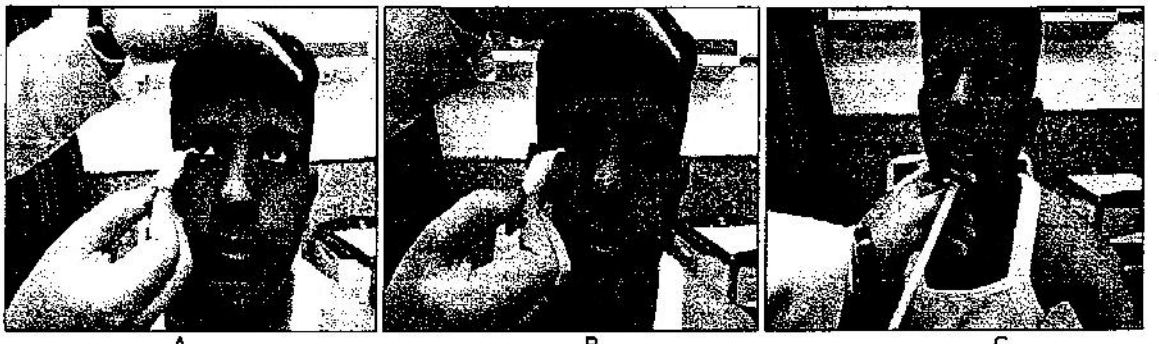
FIGURE 7 – 23. Examination of motor function of the fifth cranial nerve.

TEST CORNEAL REFLEX (FIGURE 7 – 24)

Lightly touch the cornea (not the conjunctiva) with a wisp of cotton wool brought to the eye from the side. Reflex blinking of both eyes is a normal response. The sensory component of the reflex is mediated by the ophthalmic division of the fifth nerve, while the reflex blink (motor) results from facial nerve innervation of the orbicularis oculi muscles and normally stimulating either cornea initiates a bilateral blink. The examination is repeated on the other side by reversing the directions. Those patients who are skittish about any object approaching their eyes may be tested by blowing a little breath on the eye that is tested. If there is a fifth nerve lesion, there will be a blink of both eyes when the normal side is stimulated and no response in either eye when the affected side is stimulated (afferent defect). If the seventh nerve is bilaterally involved there will be no response if either side is stimulated. There will be a blink response of the normal side when the abnormal side of the facial nerve is stimulated.

THE JAW JERK (FIGURE 7 – 24)

The afferent and efferent parts subserve by the trigeminal nerve. To evaluate the jaw jerk, place the thumb or forefinger in the midline over the tip of the patient's mandible, with the mouth slightly open. Tap your finger downwards with a tendon hammer. Normally there is a slight closure of the mouth or no reaction at all. In an upper motor neurone lesion above the pons the jaw jerk is greatly exaggerated. This is commonly seen in pseudobulbar palsy.



A B C

FIGURE 7 – 24. The technique of corneal reflex (A and B) and the jaw jerk (B).

Causes of trigeminal nerve lesion are shown in BOX 7 – 7.

BOX 7 – 7. Causes of trigeminal nerve lesion

Central (pons, medulla and upper cervical cord)	Peripheral (middle fossa)	Trigeminal ganglion	Cavernous sinus*
Vascular lesion	Aneurysm	Trigeminal neuroma	Aneurysm
Tumour	Tumour (secondary or primary)	Meningioma	Tumour
Syringobulbia	Chronic meningitis.	Fracture of the middle fossa	Thrombosis

*Cavernous sinus lesion involves ophthalmic division only and is usually associated with III, IV, and VI nerves palsies.

CRANIAL NERVE VII - FACIAL NERVE

The facial nerve innervates the muscle of facial expression. Through the nervus intermedius, it supplies parasympathetic secretomotor fibers to the lacrimal gland (producing the tears) and submandibular gland (producing saliva). It supplies taste sensation to the anterior two third of the tongue through the chorda tympani branch. It is also the efferent limb of the corneal reflex and the efferent limb of the stapedius reflex. The technique of facial nerve examination includes four plus steps:

The candidate should inspect for the wrinkles of the forehead and nasolabial folds where in the paralysed side, there may be no wrinkles or nasolabial folds. The patient is asked then to (FIGURE 7 – 25):

STEP I Raise the eye brows

STEP II Close the eyes against resistance. The patient is asked to close the eyes tightly while the examiner tries to open them. The patient is told to close the eyes as tightly as possible. The examiner can say, "Don't let me open them." Normally the examiner should not be able to open the patient's eyes

STEP III Show me your teeth

STEP IV Blow out the cheeks against resistance

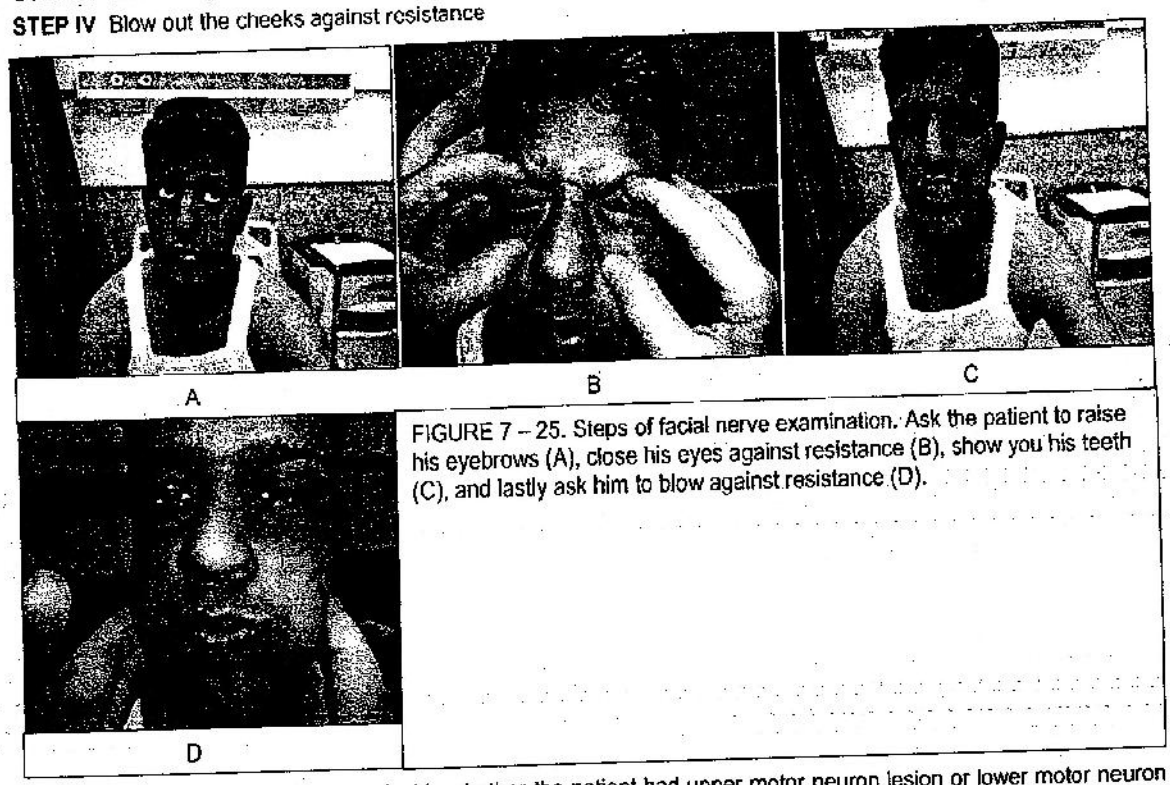


FIGURE 7 – 25. Steps of facial nerve examination. Ask the patient to raise his eyebrows (A), close his eyes against resistance (B), show you his teeth (C), and lastly ask him to blow against resistance (D).

From these tests the candidate can decide whether the patient had upper motor neuron lesion or lower motor neuron lesion (FIGURE 7 – 26). Upper motor neuron lesions produce weakness of the lower face, with normal function of the upper face. The patient thus has normal frontal wrinkling, loss of nasolabial folds, able to perform test 1 and test 2, the mouth is deviated to normal side, and unable to do test 3 and 4. This is related to the bilateral innervation of the upper face by the corticobulbar fibers. The lower face has only unilateral innervation from contralateral cortical centers.
Lower motor neuron lesions produce weakness of the total ipsilateral facial muscles. The patient will thus lose frontal wrinkling, lose of nasolabial fold, mouth deviates to normal side and becomes unable to perform the previous 4 tests.

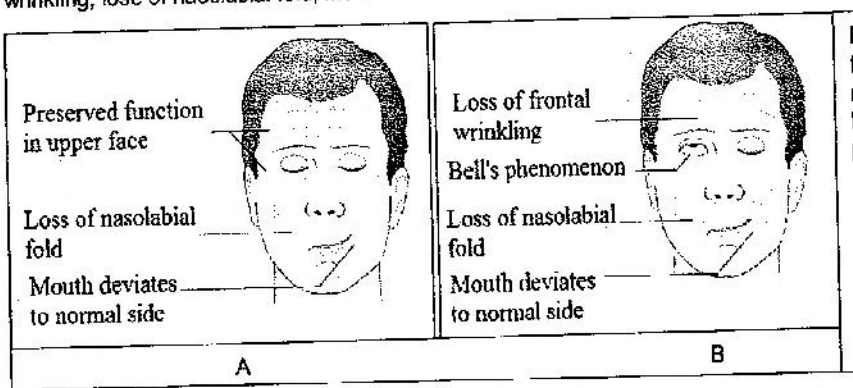


FIGURE 7 – 26. The clinical features of upper motor neuron facial palsy (A) and lower motor neuron facial palsy (B).

Bell's palsy is an idiopathic condition presenting with acute lower motor neurone VII nerve paralysis, often preceded by retroauricular pain. The inability to close the eye is often accompanied by Bell's phenomenon in which the eyeball rolls upwards. This movement is a normal response to eye closure.
Ramsay Hunt syndrome occurs when there is herpes zoster infection of the geniculate (facial) ganglion. This results in a severe lower motor neurone facial palsy and a painful vesicular eruption within the external auditory meatus.

STEP V Decide now whether the patient has upper motor neurone lesion or lower motor neuron lesion:

A Upper motor neuron lesion

Ask the patient to purse their lips (whistle for me!) and look for asymmetry (FIGURE 7 – 27). The patient will always smile after whistling. (Why this test?)

The facial nerve has two major innervations. The cortical innervation derives from the motor cortex and is bilateral to the upper facial muscles (voluntary/cortical). The seventh nerve nucleus is also innervated from the contralateral basal ganglia, thalamus and temporal lobe. This innervation and its projection to the frontal lobe constitute the "mimetic" or "emotional facial" (emotion/subcortical). It is responsible for the normal play of facial expression during speech and with laughter. If a patient laughs spontaneously, the facial muscles opposite a lesion (of the thalamus, basal ganglia or temporal lobe) do not contract. If the patient is asked to voluntarily smile, these muscles contract.

FIGURE 7 – 27. Ask the patient to whistle.

THUS

The step of "show me your teeth" is cortical and the step of "whistling" is subco

Show me your teeth	-	+	-	+
Whistling	-	-	+	+
Conclusion	Mixed	Subcortical	Cortical	Bilateral upper

B Lower motor neuron lesion

I. Check quickly for the ear and palatal vesicles of herpes zoster of the geniculate ganglion that occur in Ramsay Hunt syndrome

II. Test taste sensation

Ask the patient not to speak during the test because this will cause the tongue to retract and dissipate the substance into the contralateral side of the tongue as well as its posterior one third then ask the patient to protrude the tongue and put taste substance (usually sugar) on the affected side of the tongue

III. Test stapedius reflex

Hyperacusis can be confirmed subjectively by checking auditory function with finger rub or tuning forks and can be documented objectively by acoustic reflex testing during an audiometric evaluation

IV. Lacrimation (Schirmer's test) and saliva flow tests (both are not performed clinically)

Schirmer's test is performed by gently applying a strip of filter paper from the lower eyelids and measuring the length of moistening on each side after the patient is stimulated to tear by inhaling ammonia. Saliva flow is rarely tested. If the examiner stimulates its flow by placing a spicy substance on the tongue, which is slightly raised, the examiner can compare submaxillary flow from side to side.

SITES OF A UNILATERAL LOWER MOTOR NEURON WEAKNESS OF THE SEVENTH NERVE

If the sixth nerve is involved, the lesion is in the pons as the seventh nerve fibers wrap around the sixth nerve nucleus before they exit laterally. If the fifth and eighth nerves are involved, the lesion is in the cerebellopontine angle (CPA). It is usually a tumor with consequent ipsilateral ataxia and contralateral weakness. If taste, salivation and lacrimation are involved the lesion lies close to the brainstem prior to the bifurcation of the chorda tympani in the middle ear. The stapedius muscle is affected at this level and sounds are distorted and too loud. If taste and salivation are affected, but lacrimation is normal, the lesion is in the middle ear between the superficial petrosal nerve and the chorda tympani. If only motor weakness of the nerve is determined, the lesion is usually in the facial canal peripheral to the take off of the chorda tympani. If only some of the facial muscles are involved, particularly those innervating the upper face, the process is usually in the parotid gland or the muscles themselves.

Some causes of facial nerve palsy are provided in BOX 7 – 8.

BOX 7 – 8. Some causes of facial nerve palsy	
Unilateral upper motor nerve lesion	Bilateral upper motor nerve lesion
Stroke Tumor	Pseudobulbar palsy Motor neuron disease
Unilateral lower motor nerve lesion	Bilateral lower motor nerve lesion
Demyelination, tumours, Bell's palsy, pontine lesions, and cerebellopontine angle lesions	Sarcoidosis, Guillain Barré syndrome, and myasthenia gravis

Undergraduate students may follow these proper steps of facial nerve examination or may follow the instructions provided by their trainer during clinical training because some examiners recommend to undergraduate students to perform the five steps together without separating these belonging to upper motor neurone and these belonging to lower motor neurone.

CRANIAL NERVE VIII - VESTIBULOCOCHLEAR

The eighth cranial nerve has two components: the cochlear, with afferent fibres subserving hearing; and the vestibular, containing afferent fibres subserving balance. The sequence of examination is performed as follows:

Examination of hearing function (FIGURE 7 – 28)

1 Check the patient's ability to hear a finger rub or whispered voice with each ear while the other ear is occluded

2 Tuning fork (256-Hz) tests; Rinné's test and Weber's test (For postgraduate students)

Examination of vestibular function

1 Dix-Hallpike's test (FIGURE 7 – 28)

RINNÉ'S TEST

This test determines whether air conduction is better than bone conduction because normally the air-conducted sound is perceived as louder. The steps of examination are:

1 Mask hearing to the non-tested ear

2 Place vibrating tuning fork on the mastoid process first in order to assess bone conduction of sound then just lateral to the external ear

3 Ask the patient which of the two appears louder:

Rinné's positive (air > bone) either normal or the patient has sensorineural hearing loss
Rinné's negative (air < bone) conductive loss

WEBER'S TEST

In this test, place strongly vibrating tuning fork on the midline of the forehead then ask the patient where the sound is heard loudest. Normally the sound appears to arise in the midline. Any lateralization is abnormal:

1. In unilateral sensorineural deafness, sound is localized to the unaffected side.
2. In unilateral conduction deafness, sound is localized to the affected side.

DIX-HALLPIKE'S TEST (NON ROUTINE TEST)

The patient sits up; having warned him or her what is about to occur, the examiner grasps the patient's head between the hands and gets him or her to lie back quickly so that the head lies 30 degrees below the horizontal. At the same time, the head is rotated 30 degrees towards the examiner. Perform first with the right ear down. Perform next with the left ear down. Ask the patient to keep the eyes open. If the test is positive, after a short latent period (10 – 20 seconds) vertigo and nystagmus (rotatory) towards the affected (lowermost) ear occur for several seconds (10 – 20 seconds) and then abate (fatigue) and are not reproducible for 10 to 15 minutes. This result is seen in the condition called benign paroxysmal positioning vertigo (BPPV). If there is no latent period, no fatiguability or the nystagmus persists or is variable, this suggests that there is a lesion of the brainstem (e.g. multiple sclerosis) or cerebellum (e.g. metastatic carcinoma).

A B C

FIGURE 7 – 28. Examination of the cochlear component of the eight cranial nerve using rubbing test (A) and examination of the vestibular component by performing Dix Hallpike's test (B and C).

CRANIAL NERVES IX AND X - GLOSSOPHARYNGEAL AND VAGUS NERVES

Both nerves contain sensory, motor and autonomic components. They arise from the medulla and leave the skull together via the jugular foramen. The IX nerve passes down and forward to supply the stylopharyngeus muscle (motor), the mucosa of the posterior pharyngeal wall, the tonsillar area and the posterior third of the tongue, and sends parasympathetic fibres to the parotid gland. The X nerve courses down in the carotid sheath, receives sensory fibres from the pharynx and larynx and gives off several branches, including pharyngeal and recurrent laryngeal branches, which provide motor supply to the pharyngeal, soft palate and laryngeal muscles.
These two nerves are usually examined together. The steps of examination are:

1 Ask the patient to speak in order to assess the patient's speech for dysarthria or dysphonia (ask the patient about his/her swallowing). Dysphonia or dysarthria may result from paralysis of the vagus nerve.

2 Test the patient's ability to swallow a small amount of water and watch for regurgitation into the nose, or coughing.
Or
Ask the patient to puff out the cheeks with the lips tightly closed. Look and feel for air escaping from the nose. Normally, both sides of the palate elevate symmetrically and the uvula remains in the midline. In order for the cheeks to puff out, the palate must elevate and occlude the nasopharynx. If palatal movement is weak, air will escape audibly through the nose.

3 Ask the patient to open the mouth and inspect the palate with a torch. Note any displacement of the uvula. Then ask the patient to say 'Ah'. The soft palate normally rises symmetrically, the uvula remains in the midline, and each side of the posterior pharynx moves medially, like a curtain. The slightly curved uvula seen occasionally in a normal person should not be mistaken for a uvula deviated by a vagus nerve lesion. The palate fails to rise with a bilateral lesion of the vagus nerve. In unilateral paralysis, one side of the palate fails to rise and, together with the uvula, is pulled toward the normal side.

4 Perform gag reflex (need permission from the examiners)
Warn the patient that you are going to test the gag reflex. The candidate may use a tongue blade or an applicator stick. By touching the posterior third of the tongue, the soft palate, or the posterior pharyngeal wall, the candidate should elicit a gag reflex (ninth is the sensory component and tenth the motor component). The gag reflex initiates contraction of the pharynx, elevation of the palate, and tongue retraction. The gag reflex is performed firstly on one side and then on the other side. It may be symmetrically diminished or absent in some normal people. The most common cause of a reduced gag reflex is old age. Of more concern to the candidate is the patient with an exaggerated but still normal reflex. This can lead to vomiting!!!

5 Assess the sensation by touching the back of the pharynx on each side with a spatula (rather than the soft palate). The patient is asked if the touch of the spatula (ninth) is felt each time. Normally, there is reflex contraction of the soft palate. If contraction is absent and sensation is intact this suggests a tenth nerve palsy.

Common causes of ninth and tenth cranial nerve palsy are provided in BOX 7 – 9.

BOX 7 – 9. Common causes of IX and X nerve lesions

Unilateral of IX and X	Recurrent laryngeal	Bilateral X
Skull base tumours (meningioma) Skull base fracture Lateral medullary syndrome	Lung cancer Mediastinal lymphoma Aortic arch aneurysm Post-thyroid surgery	Progressive bulbar palsy (motor neurone disease) Bilateral supranuclear lesions (pseudobulbar palsy): • Cerebrovascular disease • Multiple sclerosis

CRANIAL NERVE XI - ACCESSORY NERVE

It has both cranial and spinal branches. The spinal accessory nerve is a motor nerve that supplies the sternomastoid and trapezius muscles. The examination of accessory nerve should include:
Ask the patient to shrug the shoulders. Feel the bulk of the trapezius muscles and attempt to push the shoulders down. Both sides should be equal. Then instruct the patient to turn the head to the side against resistance (the candidate's hand). Remember that the right sternomastoid turns the head to the left. Feel the muscle bulk of the sternomastoids. These tests are shown in FIGURE 7 – 29.
Remember that the action of the sternocleidomastoid is to turn the head to the opposite side (e.g. poor head turning to the left indicates a weak right sternocleidomastoid). Causes of accessory nerve palsy are:

1. Bilateral weakness: with wasting caused by muscular problems or motor neuron disease.
2. Unilateral weakness (trapezius and sternomastoid same side): suggests a peripheral neurological lesion.
3. Unilateral weakness (trapezius and sternomastoid of opposite sides): usually with hemiplegia suggests upper motor neurone (UMN) lesion ipsilateral to the weak sternomastoid.

FIGURE 7 – 29. Examination of the accessory nerve including trapezius muscles assessment (A) and sternocleidomastoid muscles assessment (B). Unilateral right XI palsy with droopy shoulder and associated sternocleidomastoid and trapezius atrophy in an anterior view (C) and posterior view (D).

CRANIAL NERVE XII - HYPOGLOSSAL NERVE

This nerve leaves the skull via the hypoglossal foramen and is the motor nerve for the tongue.
The examination sequences of the hypoglossal nerve (FIGURE 7 – 30) include:

1. Ask the patient to open his/her mouth with the tongue resting quietly on the floor of the mouth. Inspect for fasciculations (fine irregular non-rhythmical muscle fiber contractions) and wasting. These are indicative of a hypoglossal lower motor neuron lesion
2. Ask the patient to protrude his/her tongue. Normally, the tongue is protruded and lies in the midline. Deviation of the tongue to either side is abnormal. Because the tongue muscles push rather than pull, weakness of one side results in the tongue being pushed by the normal side to the affected side. Tremor of the resting or protruded tongue is common in Parkinson's disease
3. Ask the patient then to push the tongue against the inside of each cheek as you palpate externally for strength

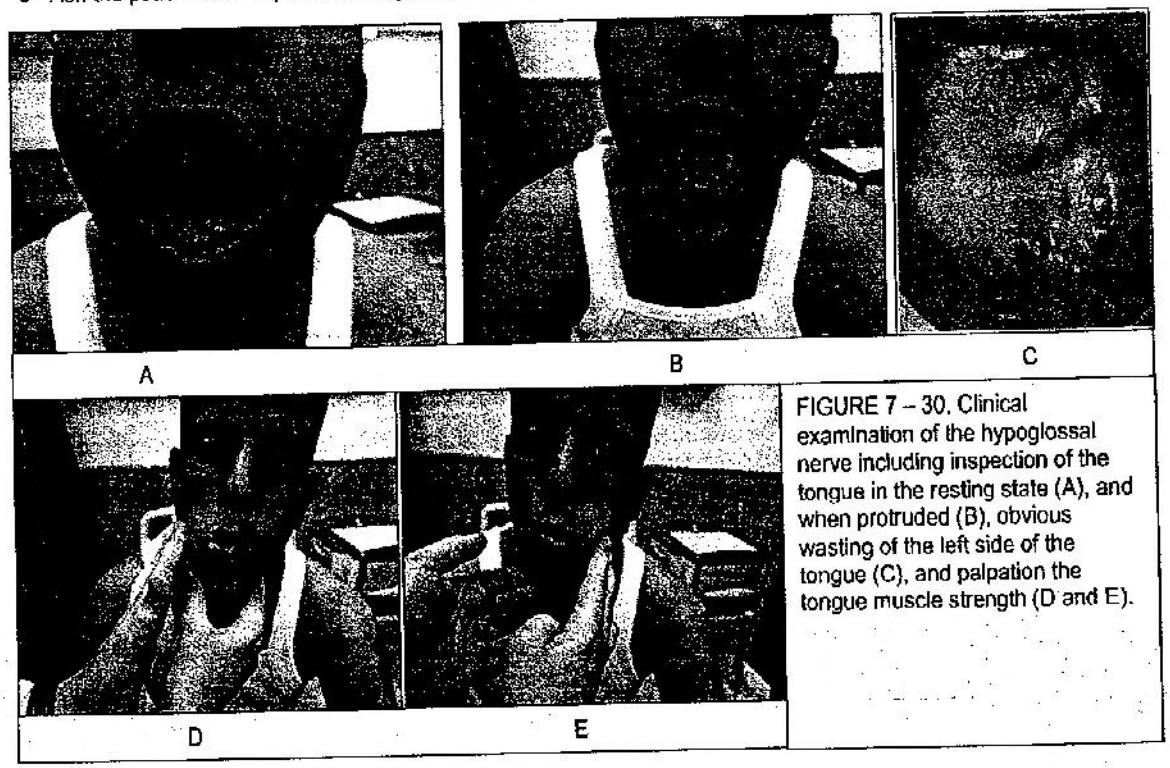


FIGURE 7 – 30. Clinical examination of the hypoglossal nerve including inspection of the tongue in the resting state (A), and when protruded (B), obvious wasting of the left side of the tongue (C), and palpation the tongue muscle strength (D and E).

Unilateral lower motor neuron lesions lead to fasciculations, wasting and weakness of the tongue on the affected side and deviation to the affected side on protrusion. Bilateral lower motor neuron damage results in global wasting and bilateral wasting. The posterior pharyngeal wall became more obvious than normal due to wasting. When associated with lesions of IX and X, typically in motor neuron disease, these features are called bulbar palsy.
Upper motor neuron lesions on one side are uncommon because the tongue, like the face and palate, has a bilateral upper motor neurone innervation in most people, while bilateral lesions lead to a spastic (the tongue is immobile and can not be protruded), bunched-up, almost conical or acorn-like tongue. The posterior pharyngeal wall became less obvious than normal due to increase in vertical tongue dimension. When associated with lesion of IX and X, these are called pseudobulbar palsy and usually result from vascular disease, motor neuron disease or occasionally multiple sclerosis.

CLINICAL
CLUE

FOR UNDERGRADUATE AND POSTGRADUATE CANDIDATES
Lesion of the seventh and tenth cranial nerves cause deviation to the normal side (pull phenomenon)
Lesion of the fifth and twelfth cranial nerves cause deviation to the affected side (push phenomenon)
This can be remembered via the following equation:
10 + 7 = 17
12 + 5 = 17

CLINICAL FEATURES OF PSEUDOBULBAR AND BULBAR PALSIES

	Pseudobulbar	Bulbar
Feature	Bilateral UMN lesions of lower cranial nerves IX, X, XI and XII	Bilateral LMN lesions of the lower cranial nerves IX, X, XI and XII
Jaw jerk	Increased	Absent or normal
Speech	Spastic dysarthria	Nasal
Emotion	Labile emotions	Normal emotions
Gag reflex	Increased or normal	Absent
Tongue	Spastic	Wasted, fasciculations
Other	Bilateral limb UMN (long tract) signs	Signs of the underlying cause- e.g. limb fasciculations
Causes	Bilateral cerebrovascular disease (e.g. both internal capsules) Multiple sclerosis Motor neurone disease	Motor neurone disease Guillain-Barré syndrome Poliomyelitis Brainstem infarction

The anatomical courses of the cranial nerves mean they can be affected in groups by single lesions that damage them when they run close to each other. Certain disease processes may also interfere with a number of the cranial nerves. There are a number of syndromes that result from abnormalities of groups of cranial nerves:

1. Unilateral III, IV, V and VI involvement — Suggests a lesion in the cavernous sinus
2. Unilateral V, VII and VIII involvement — Suggests a cerebellopontine angle lesion (usually a tumour)
3. Unilateral IX, X and XI involvement — Suggests a jugular foramen lesion
4. Weakness of the eye and facial muscles that worsens with repeated contraction — Suggests myasthenia
5. Combined bilateral IX, X and XII suggests bulbar palsy if lower motor neurone changes are present and pseudobulbar palsy if there are upper motor neurone signs

TWO TETS IN COMATOSE PATIENTS

I. Oculocephalic (doll's eye) reflex (FIGURE 7 – 31)
This reflex helps to assess brainstem function in a comatose patient. With the patient supine hold his head in both hands with your thumbs holding the eyes open so that you can see the eyes, turn the head quickly, first to one side and then to the other. Make sure the patient has no neck injury prior to performing this test. In a comatose patient with an intact brainstem, as the head is turned the eyes move toward the opposite side (Opposite = O.k.). In a patient with a lesion in the brain stem, the doll's eyes reflex is absent. The doll's eyes reflex can be elicited only in a comatose patient because alert individuals will fixate on an object and override this reflex.

FIGURE 7 – 31. Illustration of doll's eyes reflex. Remember that doll's eyes reflex can be elicited only in a comatose patient because alert individuals will fixate on an object and override this reflex.

II. Oculovestibular reflex (caloric stimulation)

If the oculocephalic reflex is absent and you seek further assessment of brainstem function, test the oculovestibular reflex. Note that this test is almost never performed in an awake patient. Make sure the eardrums are intact and the canals clear. The patient should be placed with the head flexed at 30°. This orients the semicircular canal in a horizontal position. A large-bore syringe is filled with 30 ml of ice water, and the water is squeezed into one of the external auditory canals. The normal response is the development of nystagmus. Slowly the eyes move conjugately to the ipsilateral side, followed by the rapid movement (nystagmus) of the eyes back to the midline. The use of cold water causes nystagmus to the opposite side. If warm water is used, the eyes will move rapidly toward the side being irrigated. This can easily be remembered by using the mnemonic COWS, which stands for cold opposite, warm same. Place a kidney basin under the ear to catch any overflowing water. Repeat on the opposite side, waiting three to five minutes if necessary for the first response to disappear.

BRAINSTEM SYNDROME

As the anatomy of the brain stem is very precisely organized, it is usually possible to localize the site of a lesion on the basis of careful history and examination to determine exactly which tracts/nuclei are affected as shown here:

Name of syndrome	Site of lesions	Clinical features
Weber	Anterior cerebral peduncle (mid-brain)	Ipsilateral 3rd palsy Contralateral upper motor neuron 7th palsy Contralateral hemiplegia
Claude	Cerebral peduncle involving red nucleus	Ipsilateral 3rd palsy Contralateral cerebellar signs
Parinaud	Dorsal mid-brain (tectum)	Vertical gaze palsy Convergence disorders Convergence retraction nystagmus Pupillary and lid disorders
Millard-Gubler	Ponto-medullary junction	Ipsilateral 6th palsy Ipsilateral lower motor neuron 7th palsy Contralateral hemiplegia
Wallenberg or lateral medullary syndrome	Lateral medulla	Ipsilateral 5th, 9th, 10th, 11th palsy Ipsilateral Horner's syndrome Ipsilateral cerebellar signs Contralateral spinothalamic sensory loss Vestibular disturbance

EXAMPLES

Examine the olfactory nerve of this patient
Examination of the olfactory nerve had shown that the patient had patent nasal passages and he was able to recognise the odors with no decrement or alteration in this capability.

What is the interpretation?
The interpretation is normal olfactory nerve examination.

Examination of the olfactory nerve had shown that the patient had blocked left nasal passage with nasal discharge and patent right nasal passage. The patient was not able to recognise the odors presented to the left nostril but was able to do so in his right nostril with no decrement or alteration in this capability.

What is the interpretation?
The interpretation is nasal infection.

Examination of the olfactory nerve had shown that the patient had patent right and left nasal passages. The patient was not able to recognise the odors presented to the left nostril but was able to do so in his right nostril with no decrement or alteration in this capability.

What is the interpretation?
The interpretation is left olfactory nerve or olfactory tract lesion.

Examine the optic nerve of this patient
Examination of the optic nerve of this patient had shown that the patient had normal visual acuity when examined with Rosenbaum card (Snellen chart was not available), no sensory inattention, normal central visual fields with no central scotomas, normal bilateral blind spot which extends five degrees about 20 degrees temporally, and normal peripheral visual fields with no peripheral scotomas. The patient also had normal color vision and normal ophthalmoscopic examination. Both direct and indirect light reflexes were normal.

What is the interpretation?
The interpretation is normal optic nerve examination. The light reflex is examined because the afferent arc of this reflex is conducted via the optic nerve.

Examine the visual fields of this patient
Examination of the visual fields of this patient had shown that the patient had no sensory inattention, normal central visual fields with no central scotomas, normal bilateral blind spot which extends five degrees about 20 degrees temporally, and right homonymous hemianopia.

What is the interpretation?
The interpretation is either left optic tract lesion, or left optic radiation lesion, or left occipital cortex lesion.
How can you further finely adjust your finding?
If the defect is incongruous, the lesion is located in the left optic tract.
If the defect is congruous, the lesion is located in the left optic radiation or left occipital cortex; macular sparing indicates lesion of the occipital cortex. These findings need perimetric examination.

Examine the peripheral visual fields of this patient
Examination of the peripheral visual fields of this patient had shown that the patient had right homonymous superior quadrantanopia.

What is the interpretation?
The interpretation is left temporal lobe lesion.

EXAMPLES

Examine the peripheral visual fields of this patient
Examination of the peripheral visual fields of this patient had shown that the patient had bitemporal hemianopia with the upper quadrant defects more than the lower quadrant defect.

What is the interpretation?
The interpretation is optic chiasm lesion; commonly due to pituitary tumor.

Examine the third, fourth, and sixth cranial nerves
Examination of the third, fourth, and sixth cranial nerve had shown that the patient had no ptosis, no proptosis, and the pupils were round, regular, equal in size and symmetrical in their responses. There was no nystagmus, no eye limitation, and no diplopia. The patient had normal light reflex and normal accommodation response.

What is the interpretation?
The interpretation is normal third, fourth, and sixth cranial nerves examination.

Examination of the third, fourth, and sixth cranial nerve had shown that the patient had right eye complete ptosis, no proptosis, and the pupils were round, regular, equal in size and symmetrical in their responses. There was nystagmus, eye limitation in all movements except toward the right lateral side, and diplopia in all direction except for the lateral gaze of the right side. The patient had normal light reflex and poor accommodation response.

What is the interpretation?
The interpretation is medical right third cranial nerve palsy.

Examine the fourth cranial nerve only of this patient
Examination of the fourth cranial nerve had shown that the patient had no ptosis, proptosis, and the pupils were round, regular, equal in size and symmetrical in their responses. There was right eye elevation on forward gaze that was increased with adduction and decreased with abduction, vertical nystagmus, eye limitation and diplopia when the patient looks down with the right eye down. Bielschowsky test had shown that the eye elevation was greatest when the head was tilted to the right side and decreased when the head was tilted to the left side.

What is the interpretation?
The interpretation is right fourth cranial nerve palsy.

Examine the sixth cranial nerve only of this patient
Examination of the sixth cranial nerve had shown that the patient had no ptosis, proptosis, and the pupils were round, regular, equal in size and symmetrical in their responses. The right eye was adducted on forward gaze, vertical nystagmus, eye limitation and diplopia when the patient looks to the right side laterally. The right eye could not be abducted.

What is the interpretation?
The interpretation is right sixth cranial nerve palsy.

Examine the third, fourth, and sixth cranial nerves
Examination of the third, fourth, and sixth cranial nerve had shown that the patient had no ptosis, no proptosis, and the pupils were round, regular, equal in size and symmetrical in their responses. The left eye could be abducted but could not be adducted and accompanied with right eye nystagmus. The patient had normal light reflex and normal accommodation response.

What is the interpretation?
The interpretation is left internuclear ophthalmoplegia.

EXAMPLES

Examine the trigeminal nerve of this patient
Examination of the trigeminal nerve had shown that the patient had normal facial sensation, normal, no wasting of the temporalis or the masseter muscles, and no jaw deviation. The patient had normal corneal reflex and normal jaw jerk.

What is the interpretation?
The interpretation is normal trigeminal nerve examination.

Examination of the trigeminal nerve had shown that the patient had no facial sensation in all the three branches of the right face, wasting of the right temporalis and the masseter muscles, and jaw deviation toward the left side. The patient had normal bilateral corneal reflex when the left cornea was stimulated, absent bilateral corneal reflex when the right cornea is stimulated, and absent jaw jerk.

What is the interpretation?
The interpretation is right trigeminal nerve palsy.

Examine the facial nerve of this patient
Examination of the facial nerve had shown that the patient had normal frontal wrinkles and nasolabial folds, capable to raise both eye brows and to close the eyes against resistance. He was also able to blow his cheeks fully against resistance and having no deviation on showing his teeth.

What is the interpretation?
The interpretation is normal facial nerve examination.

Examination of the facial nerve had shown that the patient had preserved forehead wrinkles bilaterally but absent right nasolabial folds, capable to raise both eye brows and to close the eyes against resistance. The patient's mouth is deviated to the left side. The patient was not able to blow his cheeks fully against resistance as the air is escaped and having left sided deviation on showing his teeth. Upon smiling, the patient's mouth deviation was corrected.

What is the interpretation?
The interpretation is right UMN facial nerve palsy. The lesion is cortical in origin.

Examination of the facial nerve had shown that the patient had absent forehead wrinkles and nasolabial folds on the right side, unable to raise the right eye brows and also unable to close the right eye with bell's phenomenon. The patient's mouth is deviated to the left side. The patient was not able to blow his cheeks fully against resistance as the air is escaped and having left sided deviation on showing his teeth. The patient had no external ear vesicles and tests sensation was impaired on the right side of the tongue. He heard loud voice on the right ear on finger rubbing.

What is the interpretation?
The interpretation is right LMN facial nerve palsy.
What other tests you would like to perform?
I would like to perform Schirmer's test and sallivary flow tests.
What other cranial nerves you would like to examine?
I would like to examine the fifth, sixth, and the eight cranial nerves.

Examine the eight cranial nerve of this patient
Examination of the eighth cranial nerve had shown that the patient had poor hearing ability on rough assessment of the right ear with finger rubbing.

What other tests you would like to perform in fully assessing the eighth cranial nerve?
I would like to perform Rinné's test, Weber's test, and Dix-Hallpike's test.

EXAMPLES

What other cranial nerves you would like to examine? Why?
I would like to examine the fifth and the seventh cranial nerve to detect cerebellopontine lesion.

Examine the ninth and the tenth cranial nerves of this patient
Examination of the ninth and the tenth cranial nerves had shown that the patient had no dysarthria, no dysphonia, and no dysphagia. The soft palate was centrally located in the midline and symmetrically raised on saying Ah; each side of the posterior pharyngeal wall moves medially. The patient had intact posterior pharyngeal wall sensation and intact gag reflex.

What is the interpretation?
The interpretation is normal ninth and tenth cranial nerves examination.

Examination of the ninth and the tenth cranial nerves had shown that the patient had nasal speech, dysphonia, and oropharyngeal dysphagia. The uvula was deviated to the left side; the right side of the soft palate failed to rise on saying Ah; the right side of the soft palate and the uvula is pulled toward the left side. The patient had intact posterior pharyngeal wall sensation and absent gag reflex.

What is the interpretation?
The interpretation is right tenth cranial nerve lesion.

Examination of the ninth and the tenth cranial nerves had shown that the patient had nasal speech, dysphonia, and oropharyngeal dysphagia. The uvula was deviated to the left side; the right side of the soft palate failed to rise on saying Ah; the right side of the soft palate and the uvula is pulled toward the left side. The patient had impaired posterior pharyngeal wall sensation on the right side and absent gag reflex; the left side elicited normal sensation.

What is the interpretation?
The interpretation is right ninth and tenth cranial nerves lesion.

Examination of the ninth and the tenth cranial nerves had shown that the patient had nasal speech, dysphonia, and oropharyngeal dysphagia. The uvula was centrally located in the midline; the soft palate was bilaterally failed to rise on saying Ah. The patient had exaggerated gag reflex with absence of posterior pharyngeal wall sensation bilaterally.

What is the interpretation?
The interpretation is bilateral UMN ninth and tenth cranial nerves lesion (pseudobulbar palsy).

Examine the eleventh cranial nerve of this patient
Examination of the eleventh cranial nerve of this patient had shown that the patient had normal bulk and strength of both the trapezius and sternocleidomastoid muscles bilaterally; both sides were equal and symmetrical and the patient was able to turn his head smoothly symmetrically.

What is the interpretation?
The interpretation is normal accessory nerve examination.

Examination of the eleventh cranial nerve of this patient had shown that the patient had bilateral weakness and wasting of both the trapezius and sternocleidomastoid muscles; the patient was unable to raise his head from the pillows neither he was able to turn his head bilaterally.

What is the interpretation?
The interpretation is bilateral accessory nerves lesion e.g. motor neurone disease (MND).

EXAMPLES

Examination of the eleventh cranial nerve of this patient had shown that the patient had right sided weakness and wasting of both the trapezius and sternocleidomastoid muscles; the patient was unable to turn his head to the left side.

What is the interpretation?
The interpretation is peripheral neuropathy.

Examination of the eleventh cranial nerve of this patient had shown that the patient had left sided weakness and wasting of the trapezius and weak right sternocleidomastoid muscles; the patient was unable to turn his head to the left side.

What is the interpretation?
The interpretation is right accessory nerves lesion e.g. stroke.

Examine the hypoglossal nerve of this patient
Examination of the hypoglossal nerve of this patient had shown that the patient had normal tongue contour in the resting state with no wasting and no fasciculation; the tongue is protruded smoothly in the midline with no tremor or deviation. The patient had normal strength of both sides of the tongue.

What is the interpretation?
The interpretation is normal hypoglossal nerve examination.

Examination of the hypoglossal nerve of this patient had shown that the patient had wasting and fasciculations of the right side of the tongue in the resting state inside the mouth; the tongue is deviated to the right side on protrusion. The patient had poor muscle strength of the right side of the tongue with normal strength of the left side.

What is the interpretation?
The interpretation is right LMN hypoglossal nerve lesion.

Examination of the hypoglossal nerve of this patient had shown that the patient had bilateral wasting and fasciculations of the tongue in the resting state inside the mouth; the patient had poorly protruded his tongue; the posterior pharyngeal wall is more obvious because the vertical height of the tongue is reduced. The patient had poor muscle strength of the tongue bilaterally.

What is the interpretation?
The interpretation is bilateral LMN hypoglossal nerve lesion e.g. bulbar palsy.

Examination of the hypoglossal nerve of this patient had shown that the patient had conical bunched-up tongue; the patient was not able to protrude his tongue because the tongue was immobile; the posterior pharyngeal wall was less obvious than usual because the vertical height of the tongue is increased. The patient could not push his tongue against the inside checks bilaterally.

What Is the interpretation?
The interpretation is bilateral UMN hypoglossal nerve lesion e.g. pseudobulbar palsy.

MENINGEAL SIGNS

These signs (FIGURE 7 – 32) should be looked for in patient in whom meningitis or subarachnoid hemorrhage is a possibility; however, these may be absent in the early evolution of a subarachnoid hemorrhage or in the deeply comatose patient

NECK STIFFNESS

It describes the limitation of the passive neck forward movement. First make sure there is no injury to the cervical vertebrae or cervical cord. Then, with the patient supine, place your hands behind the patient's head and turn the head laterally in both direction and then flex the neck forward passively (i.e. without assistance from the patient), until the chin touches the chest if possible. Normally the neck is supple and the patient's head and neck can be bent forward. Pain in the neck and resistance to flexion can arise from a number of conditions provoking painful extensor muscle spasm including bacterial and viral meningitis, subarachnoid hemorrhage, Parkinsonism, raised intracranial pressure, cervical spondylosis, cervical lymphadenopathy, and pharyngitis. In Parkinsonism and in cervical spondylitis, the limitation is present in all directions of movement, in distinction to that of meningitis, which is present only or predominantly on forward flexion. Whether it is stiffness in the initial few degrees of flexion of the neck or in the subsequent part of the movement that is more specific for meningitis has been debated; it has been suggested that the latter (terminal catch) is more sensitive but also proves to be mistaken for other disorders; therefore the first (initial catch) may be more specific for meningitis.

KERNIG'S SIGN

Flex the patient's leg at both the hip and the knee, and then straighten the knee. Discomfort behind the knee during full extension occurs in many normal people, but this maneuver should not produce pain. Kernig's sign if positive, there will be resistance to leg straightening (caused by hamstring spasm as a result of inflammation around the lumbar spinal roots) and pain felt at the back of the neck. When bilateral, it suggests meningeal irritation.

BRUDZINSKI'S SIGN

As you flex the neck, watch the hips and knees in reaction to your maneuver. Normally they should remain relaxed and motionless. Flexion of the hips and knees indicates a positive Brudzinski's sign and suggests meningeal inflammation.

A B C

FIGURE 7 – 32. Meningeal signs including testing for neck stiffness (A) and Kernig's sign (B and C).

CLINICAL CLUE

Kernig's and Brudzinski's signs may be absent or reduced in very young or elderly patients, in patients with a severely depressed mental status, and in immunocompromised individuals.

The term meningism (meninglsmus, nuchal rigidity) describes the stiffness or discomfort on passive movement (especially flexion) of the neck in the presence of meningeal irritation.

MOTOR SYSTEM EXAMINATION

The motor system examination includes the following steps:

1 General inspection and palpation
2 Muscle tone
3 Muscle strength (power)
4 Reflexes
5 Cerebellar coordination

INSPECTION AND PALPATION

This step should include body position, involuntary movements, and muscle bulk

I. BODY POSITION

Observe the patient's body position during movement and at rest. Abnormal positions alert you to neurologic deficits such as paralysis. In decorticate posture (abnormal flexor response), the upper arms are held tight to the sides with elbows, wrists, and fingers flexed. The legs are extended and internally rotated. The feet are plantar flexed. This posture (FIGURE 7 – 33) implies a destructive lesion of the corticospinal tracts within or very near to the cerebral hemispheres. When unilateral, this is the posture of chronic spastic hemiplegia. In decerebrate posture (abnormal extensor response), the jaws are clenched and the neck is extended. The arms are adducted and stiffly extended at the elbows, with forearms pronated, wrists and fingers flexed. The legs are stiffly extended at the knees, with the feet plantar flexed. This posture (FIGURE 7 – 33) may occur spontaneously or only in response to external stimuli such as light, noise, or pain. It is caused by a lesion in the diencephalon, midbrain, or pons, although severe metabolic disorders such as hypoxia or hypoglycemia may also produce it.

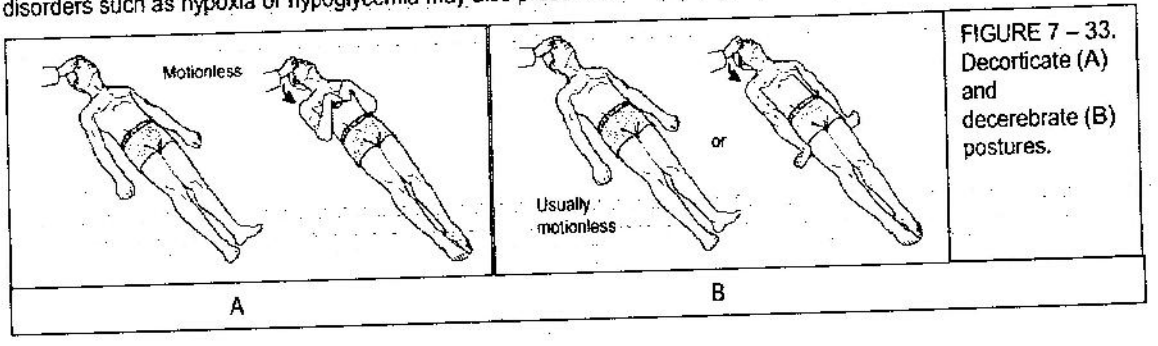


FIGURE 7 – 33. Decorticate (A) and decerebrate (B) postures.

II. INVOLUNTARY MOVEMENT

Watch for involuntary movements such as tremors, tics, fasciculations, etc. Note their location, quality, rate, rhythm, and amplitude, and their relation to posture, activity, fatigue, emotion, and other factors.

A tremor is a rhythmic oscillating movement of a limb or part of a limb, or of the head. Tremors may be roughly subdivided into four groups: resting (or static) tremors, postural tremors (fine, coarse, familial), intention (cerebellar), and flapping tremors. The other characteristic by which tremors can be classified is their frequency. Rest tremor, at a frequency of 4–6 Hz, is characteristic of Parkinson's disease. The tremor typically appears unilaterally, first distally, involving the digits and wrist, where it may have a "pill-rolling" character. Tremor usually spreads proximally and occasionally to the ipsilateral leg before appearing on the other side after a year or more. It may appear later in the lips, tongue, and jaw but spares the head and neck. This tremor is most prominent at rest, and may decrease or disappear with voluntary movement. Less common causes of rest tremor include poisoning with heavy metals like mercury and Wilson disease. Postural tremor appears when the affected part is actively maintaining a posture. Examples include the fine (physiological) tremor, coarse (enhanced physiological), and benign essential tremor. The fine tremor, at frequency of 8–12 Hz, can be identified in the limbs of normal subjects. Coarse tremor can be seen in association with fear, anxiety, excessive physical activity, sleep deprivation, drugs (lithium, tricyclic antidepressants, and bronchodilators), and thyrotoxicosis. Familial tremor resembles physiological tremor, but is slower and may become quite disabling. The condition is often familial, and in some families the tremor is most obvious during certain specific actions such as writing; here there is an overlap with focal dystonias. Characteristic of essential tremor is that alcohol suppresses it, sometimes to the extent that the patient becomes addicted. Intention tremor, at frequency of 2–4 Hz, is the characteristic oscillation at the end of a movement which occurs in cerebellar disease. Flapping tremor, asterixis, is the result of intermittent failure of the parietal mechanism required to maintain a posture. Occasionally, unilateral asterixis can be seen in an acute parietal lesion, usually vascular. Fasciculation looks like irregular ripples or twitches under the skin overlying muscles at rest. They are described as irregular, non-rhythmic contractions of the muscle unit caused by spontaneous firing of anterior horn cells or their axons. These movements do not displace a joint, are coarse or fine, and are most easily seen in the deltoid, quadriceps and calves although they can occur in any

muscle group. Fasciculations are due to lower motor neurone disease (usually in wasted muscles), motor root compression, peripheral neuropathy e.g. diabetic, and in primary myopathy. The closer the lesion is to the anterior horn cell the more fasciculations are noted. They are present at rest and suppressed with voluntary movement. They are facilitated by heat or provoked by mechanically stimulating the muscle (flick the skin over wasted muscle to elicit fasciculation). Non-pathological fasciculation is common in healthy people and not associated with weakness or wasting. Chorea (BOX 7 – 10) describes rapid, jerky movements which often move from one part of the body to another, no two of which are the same, which are often described as semipurposeful. They are associated with irregular respiration, rapid protrusion and retraction of the tongue with a peculiar flapping of the tip. The small joints of the hand are hyperextended and bizarre postures of the fingers, toes and wrist are noted. The patient is unable to hold the arms and hands palm forward above the head, because the hands start to pronate. The tongue cannot be held out of the mouth. Slower, sinuous movement of the limb is termed athetosis. Chorea is caused by damage to the basal ganglia especially the caudate nucleus.

BOX 7 – 10. Causes of chorea

Hereditary		Endocrine	
Huntington's disease	Wilson's disease	Pregnancy	Hypoparathyroidism
Cerebral birth injury (including kernicterus)		Thyrotoxicosis	Hypoglycemia
Cerebral trauma		Infective/inflammatory	
Drugs		Post-streptococcal (Sydenham's chorea)	Antiphospholipid antibody syndrome
Levodopa	Tricyclic antidepressants	Henoch-Schönlein purpura	SLE
Dopamine agonists	Oral contraceptive		
Phenothiazines			

Hemiballism occurs following damage to the subthalamic nucleus. Ballistic movements are involuntary, sudden, jerking movements which occur contralateral to the lesion. The ballistic movement primarily affects the proximal limb musculature whilst the distal muscle may display more choreiform – like movements. Symptoms may decrease whilst the patient is a sleep. Myoclonic jerks are sudden shock-like contractions of one or more muscles which may be focal or diffuse and occur singly or repetitively. Healthy individuals commonly experience these when falling asleep.

Dystonia consists of sustained or repetitive involuntary muscle contractions, frequently causing twisting movements with abnormal postures and sometimes tremor. Dystonia can be classified based on age of onset (childhood vs. adult), distribution (focal, multifocal, segmental, or generalized), or etiology (primary or secondary). Secondary dystonia occurs due to neuroleptic drugs or other neurologic problems. Focal dystonias are the most common forms of dystonia and include blepharospasm (dystonic contractions of the eyelids with increased blinking that can interfere with reading, watching TV, and driving), cervical dystonia, and limb dystonia. In cervical dystonia, there is dystonic contraction of neck muscles, causing the head to deviate to one side (torticollis), in a forward direction (anterocollis), or in a backward direction (retrocollis). Limb dystonias can be present in either arms or legs and are often brought out by task-specific activities such as handwriting (writer's cramp). Myokymia describes continuous quivering and rippling movements of muscles at rest like a bag or worms. Myokymia is related to myotonia and stiffness, since there may be concurrent impairment of muscle relaxation and a complaint of muscle cramps. Akathisia is a feeling of inner restlessness, often associated with restless movements of a continuous and often purposeless nature, such as rocking to and fro, repeatedly crossing and uncrossing the legs, standing up and sitting down, and pacing up and down. Recognized associations of akathisia include Parkinson's disease and neuroleptic medication use. Tics are repetitive semi-purposeful movements such as blinking, winking, grinning or screwing up of the eyes. They are distinguished from other involuntary movements by patient's ability to suppress it, at least for a short time.

iii. Muscle Bulk (FIGURE 7 – 34)

Inspect and palpate muscles to assess their bulk (normal, atrophied, or hypertrophied). Muscular atrophy refers to a loss of muscle bulk (wasting) and the muscles look flat or concave; hypertrophy refers to an increase in bulk with proportionate strength, while increased bulk with diminished strength is called pseudohypertrophy. Wasted muscles feel 'flabby'; inflamed muscles (myositis) may be tender; and some forms of acute muscle necrosis (rhabdomyolysis) produce a firm 'woody' feel. Certain occupations and sports lead to muscle hypertrophy. Pseudohypertrophy occurred in muscular dystrophy gives a 'doughy' feeling on palpation which differs from healthy hypertrophic muscles. Lower motor neurone lesions cause muscle wasting. Wasting is not seen in acute upper motor neurone lesions but atrophy may develop with longstanding lesions. Determine then whether the atrophy is unilateral or bilateral, proximal or distal, and focal or global (generalized). When looking for atrophy, pay a particular attention to the hands, shoulders, and thighs. The thenar and hypothenar eminences should be full and convex, and the spaces between the metacarpals, where the dorsal interosseous muscles lie, should be full or only slightly depressed. Muscular atrophy can be assessed by comparison of left limb with the right limb or upper limb with the lower limb so that atrophied or wasted muscle appears smaller. In general, proximal weakness is related to muscle disease (except myotonia dystrophica, in which it is distal); distal weakness is related to neurologic disease. Certain forms of muscular dystrophy involve

weakness of the pelvic girdle muscles. Children with this disease rise from a supine to a standing position in a characteristic manner by rolling over to a prone position, pushing off the floor with the arms, bringing the legs to a flexed position under the trunk, and extending the legs with help of the hands (Gower's sign).

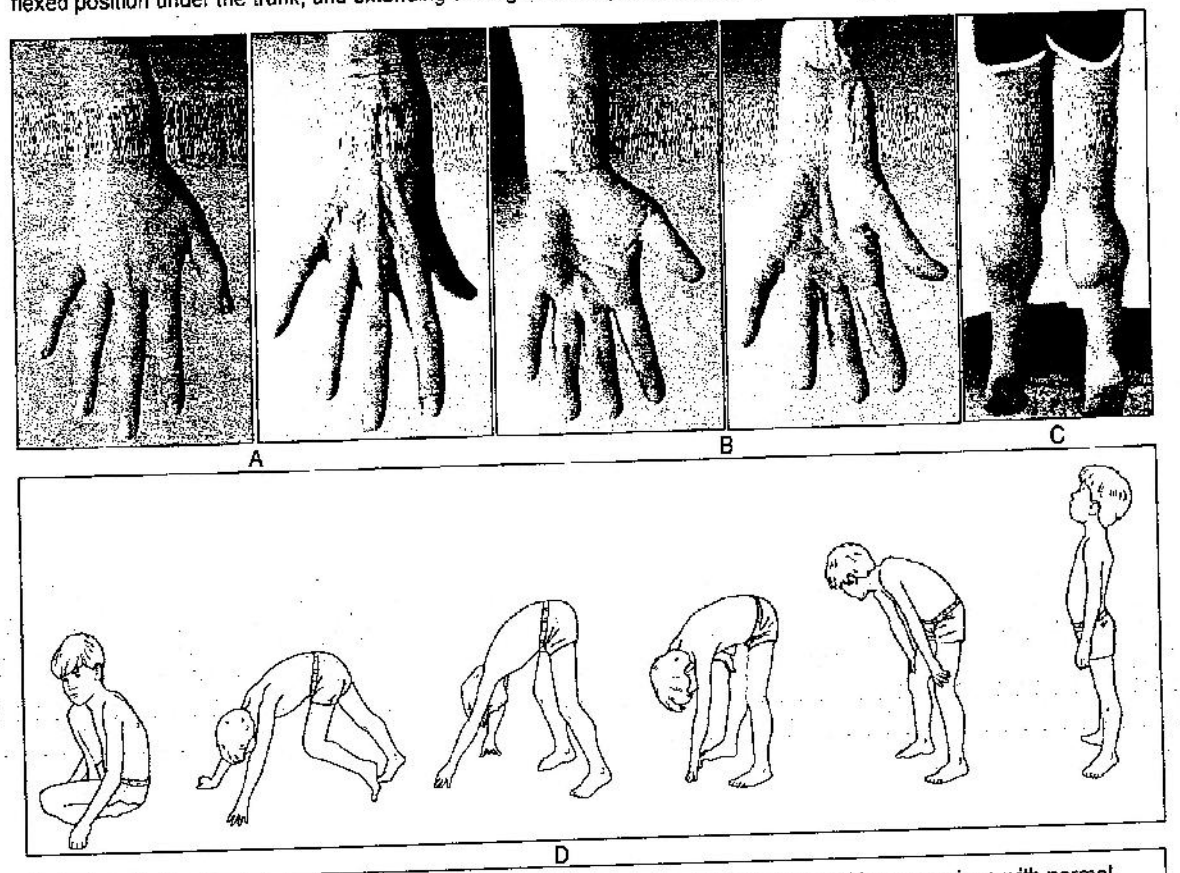


FIGURE 7 – 34. Neurologic signs including wasting of the dorsal surface of the hand in comparison with normal hand (A), thenar and hypothenar wasting in comparison with normal palm (B), pseudohypertrophy of the calf muscles (C), and Gower's sign (D).

CLINICAL
CLUE

During examination try to elicit fasciculation if they are not obvious by flicking the deltoid muscles in the upper limbs and the inner thighs and the calves in the lower limbs

MUSCLE TONE

Tone can be defined as the resistance of muscle felt by the examiner to passive movement of a joint. To assess resistance to passive movement, the patient is asked to relax while each limb is examined in turn by passively taking the major joints into their full range of movement at different speeds i.e. tone is tested slowly at first and then more rapidly to get a feel of muscle tension. Compare one side with the other. The sequence of examination includes:

UPPER LIMBS EXAMINATION: test muscle tone at finger joints, wrist, elbow, and shoulder joints (FIGURE 7 – 35)

1 Ask the patient to relax. Take one hand with yours and, while supporting the elbow, flex and extend the patient's fingers; supinate and pronate the hand; rotate, flex and extend the patient's wrist; flex and extend the patient's elbow, and put the shoulder through a moderate range of motion. With practice, these actions can be combined into a single smooth, movement. Compare each side with the contralateral one.
2 If you suspect decreased resistance, hold the forearm and shake the hand loosely back and forth. Normally the hand moves back and forth freely but is not completely floppy.
3 If resistance is increased, determine whether it varies as you move the limb or whether it persists throughout the range of movement and in both directions, for example, during both flexion and extension
4 If you suspect cogwheel rigidity ask the patient to open and close the other hand (reinforcement) while you rotate the wrist joint. This often accentuates or brings out the sign.

FIGURE 7 – 35. Clinical examination of upper limb tone including flexion (A) and extension (B) of finger joints; flexion (C) and extension (D) of wrist joint; flexion (E) and extension (F) of elbow joints; adduction (G) and abduction (H) of shoulder joints; reinforcement of wrist joint (I). The patient is asked to open and close his left hand for reinforcement while the candidate rotates the wrist joint to elicit cog-wheel rigidity.

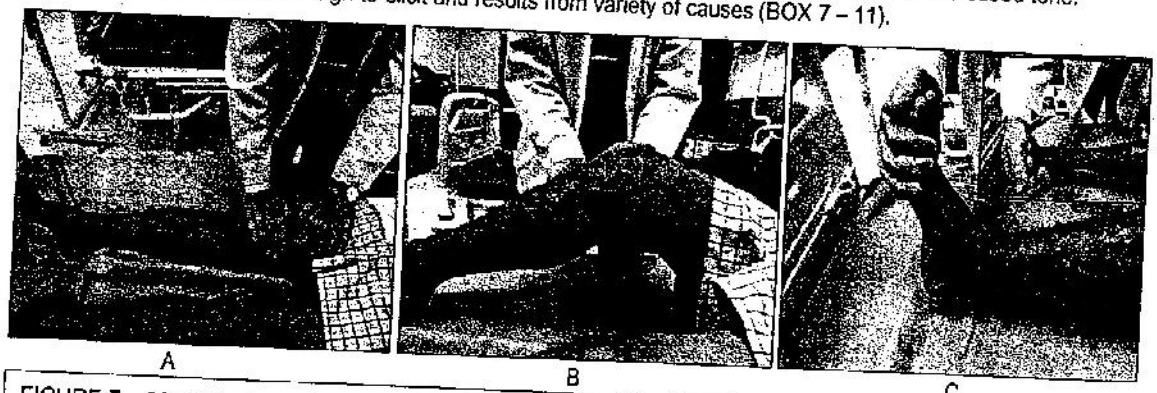
LOWER LIMBS EXAMINATION: test muscle tone at both the knee and ankle joints (FIGURE 7 – 36)

1. Place both your hands over the right thigh of the patient; rotate it internally and externally (rock it to and fro) then place both your hands under knee; raise it upwards then briskly twice or thrice. Then place the left hand under the knee; raise it while your right hand grasping foot to rotate the ankle joint
2. If you suspect decreased resistance, hold the leg and shake it loosely back and forth whilst you look at the foot. Normally the foot moves back and forth freely but is not completely floppy.
3. If resistance is increased, determine whether it varies as you move the limb or whether it persists throughout the range of movement and in both directions, for example, during both flexion and extension

Abnormalities in the tone may take the form of hypertonia (increased tone), hypotonia (reduced tone), paratonia, or myotonia. Two types of hypertonia (increased tone) can be clinically distinguished: spasticity and rigidity. Spasticity is described as a one-way resistance to extension in the arm or to flexion in the leg. Spasticity is maximal at the beginning of the extension (the "clasp-knife" phenomenon) or with faster stretch. Rigidity is increased tone that is present throughout the range of motion (a "lead pipe" or "plastic" stiffness) in all directions i.e. it affects flexors and extensors equally; it sometimes has a cogwheel quality. Cog-wheel rigidity is caused by superimposed tremor. Cog-wheel rigidity is most easily noted at the wrist when rotated. Opening and closing the opposite hand ("reinforcement") often accentuates or brings out this sign.

A lesion of the upper motor neuron whether at its origin or in the spinal cord produces spasticity. Rigidity of the lead pipe or plastic type occurs with all forms of parkinsonian syndromes. Cog – wheel rigidity also occurs in all forms of

parkinsonian syndromes but is most helpful in Parkinson's disease in the early stages. Extrapyramidal rigidity in a slight form is a concomitant of aging. Joint and muscle contractures can give the impression of increased tone. Hypotonia is a difficult clinical sign to elicit and results from variety of causes (BOX 7 – 11).



A B C

FIGURE 7 – 36. Clinical examination of lower limb tone includes rocking the thigh to and fro (A), raising the knee to check the full range of movement (B), and examining the ankle joint (C).

BOX 7 – 11. Causes of hypotonia
Lesion of the muscle itself
Lesions of the sensory side of the reflex arc e.g. sensory neuropathies. Large fibers neuropathy decreases tone more than small fiber neuropathy
Lesions of the motor side of the reflex arc e.g. lower motor neuron disease such as poliomyelitis, and primary spinomuscular atrophy, GBS and motor peripheral neuropathies
Combined motor and sensory lesions e.g. syringomyelia, cord or root lesions
Spinal or cerebral shock such as large internal capsule strokes or hemorrhage
Cerebellar lesions
Chorea e.g. Sydenham's and Huntington's chorea
The collagen disorders e.g. Marfan's disease, osteogenic imperfecta, and Ehlers–Danlos syndrome that are associated with hyperextensibility of distal extremity joints and hypotonia

Some patients give the impression of being unable to relax and will move the limb being examined as the candidate moves it, despite instructions to the contrary. In more advanced cases, there seems to be rigidity when the candidate moves the limb rapidly but normal tone when the limb moves slowly. This phenomenon (paratonia) is particularly apt to occur in patients with frontal lobe or diffuse cerebral disease.

Myotonia is an abnormality of tone that is worse after active movement. In these patients, tone is usually normal at rest but after sudden movements there may be a great increase in tone and the patient is unable to relax the muscle. Myotonia is best tested (FIGURE 7 – 37):

A B C

FIGURE 7 – 37. Examination of myotonia includes asking the patient to make hand fist and then to open his hand (A), percussion of thenar eminence (B), and testing for tongue myotonia by placing the extruded tongue on tongue blade and then tapping it (C). In each test the candidate should look at the slow relaxation of the muscles.

1 By asking the patient to make a tight fist and then open the hand quickly. When the muscles are myotonic the opening of the fist is very slow

2 Percussion of the thenar eminence causes adduction of the thumb and persistent dimpling of the muscle
3 Myotonia of the tongue can be demonstrated by having the patient place the extruded tongue on a tongue blade and then place a vertical tongue blade on the tongue which is then tapped. The indentation that occurs from the myotonic contraction may last for several seconds
4 Patient with facial myotonia cannot open the eyes following a cough

The myotonias are categorized as those with dystrophic features and those without. The classic example of myotonia with dystrophy is myotonic dystrophy which is a group of inherited disorders characterised by progressive degeneration of groups of muscles, sometimes with involvement of the heart muscle or conducting tissue, and other parts of the nervous system. Wasting and weakness are usually symmetrical, there is no fasciculation and no sensory loss, and tendon reflexes are preserved until a late stage, except in myotonic dystrophy (BOX 7 – 12).

BOX 7 – 12. Some types of muscular dystrophies

Type	Age of onset	Muscles affected	Other features
Myotonic dystrophy (DM1)	Any	Face (incl. ptosis), stemomastoids, distal limb, generalised later	Myotonia, cognitive dulling, cardiac conduction abnormalities, lens opacities, frontal balding, hypogonadism
Proximal myotonic myopathy (DM2)	Adult	Proximal (thigh), sometimes muscle hypertrophy	As for DM1 but cognition not affected Muscle pain
Duchenne	First 5 years	Proximal and limb girdle	Pseudohypertrophy of calves and cardiomyopathy
Becker	Late childhood/ early adult	Proximal and limb girdle	Pseudohypertrophy of calves and cardiomyopathy
Limb girdle	Childhood/early adult	Limb girdle	Some have calf hypertrophy Some have cardiac conduction abnormalities
Facioscapulohumeral dystrophy (FSHD)	7-30 years	Face and upper limb girdle	Pain in shoulder girdle common

Myotonia without dystrophy includes a group of disorders called channelopathies (BOX 7 – 13).

BOX 7 – 13. Some groups of channelopathies

Channel	Muscle disease	Clinical features
Sodium	Paramyotonia congenita	Cold-evoked myotonia with episodic weakness provoked by exercise and cold
	Hyperkalemic periodic paralysis	Brief, frequent episodes of weakness triggered by potassium ingestion
	Hypokalemic periodic paralysis	Episodic weakness triggered by carbohydrate meal
Chloride	Myotonia congenita (Thomsen's disease)	Generalised myotonia with muscle hypertrophy

MUSCLE STRENGTH (POWER)

Normal individuals vary widely in their strength according to age, sex, and muscular training. A person's dominant side is usually slightly stronger than the other side. Keep this difference in mind when you compare sides.
The examination sequence should follow the following steps:

1 Test muscle strength by asking the patient to move actively against your resistance or to resist your movement. Remember that a muscle is strongest when shortest, and weakest when longest
2 If the muscles are too weak to overcome resistance, test them against gravity alone by asking the patient to raise that part against gravity. When the forearm rests in a pronated position, for example, dorsiflexion at the wrist can be tested against gravity alone
3 If the muscle are weak against gravity test them without gravity by asking the patient to slide the arm or the leg laterally
3 Finally, if the patient fails to move the body part, watch or feel for weak muscular contraction

Impaired muscle strength is called weakness (paresis). Absence of strength is called paralysis. Hemiparesis refers to weakness of one half of the body; hemiplegia to paralysis of one half of the body; paraplegia means paralysis of the legs; quadriplegia means paralysis of all four limbs. Muscle strength is traditionally graded using the scale shown in BOX 7 – 14.

BOX 7 – 14. Muscle strength scale

0	No movement
1	Flicker or trace of contraction but no associated movement at a joint
2	Movement with gravity eliminated
3	Movement against gravity but not against resistance
4 –	Movement against a mild degree of resistance
4	Movement against moderate resistance
4 +	Movement against strong resistance
5	Full power

CLINICAL CLUE

Undergraduate students probably should only understand the technique of the power examination while postgraduate students should know the muscles and nerve roots in addition.

THE HAND (FIGURE 7 – 38)

I. Test finger flexion (finger grip)

The patient is asked to grasp your extended index and middle fingers and to squeeze them as hard as possible. It is important for the candidate to remove any rings, which may produce discomfort.

THE MUSCLE(S)	NERVE ROOT(S)
Flexor digitorum profundus and sublimis	(C7, C8)

II. Test finger abduction

Ask the patient to extend the hand and to spread the fingers as widely as possible. Tell the patient to resist your attempt to bring the fingers together.

THE MUSCLE(S)	NERVE ROOT(S)
Dorsal interossei	(C8, T1)

A B

FIGURE 7 – 38. Clinical examination of finger flexion (A) and abduction (B).

THE WRIST (FIGURE 7 – 39)

I. Test wrist extension

Instruct the patient to make a fist and extend the wrist while you attempt to push it down.

THE MUSCLE(S)	NERVE ROOT(S)
Extensor carpi group	(C7, C8)

II. Test wrist flexion

The patient is asked to make a fist and flex the wrist while you attempt to pull it up.

THE MUSCLE(S)	NERVE ROOT(S)
Flexor carpi ulnaris and radialis	(C6, C7)

A B

FIGURE 7 – 39. Clinical examination of wrist flexion (A) and extension (B).

THE ELBOW (FIGURE 7 – 40)

I. Test elbow flexion

The patient should bend the elbow and pull so as not to let the candidate straighten it out.

THE MUSCLE(S)	NERVE ROOT(S)
Biceps and brachialis	(C5, C6)

II. Test elbow extension

The patient should bend the elbow and push so as not to let the examiner bend it.

THE MUSCLE(S)	NERVE ROOT(S)
Triceps brachii	(C7, C8)

A B

FIGURE 7 – 40. Clinical examination of elbow flexion (A) and extension (B).

THE SHOULDER (FIGURE 7 – 41)

I. Test shoulder abduction

The patient should abduct the arms with the elbows flexed and resist the examiner's attempt to push them down.

THE MUSCLE(S)	NERVE ROOT(S)
Mostly deltoid and supraspinatus	(C5, C6)

II. Test shoulder adduction

The patient should adduct the arms with the elbows flexed and not allow the examiner to separate them.

THE MUSCLE(S)	NERVE ROOT(S)
Mostly pectoralis major and latissimus dorsi	(C6, C7, C8)

A B

FIGURE 7 – 41. Clinical examination of shoulder adduction (A) and abduction (B).

THE HIP (FIGURE 7 – 42 and 7 – 43)

I. Test hip flexion

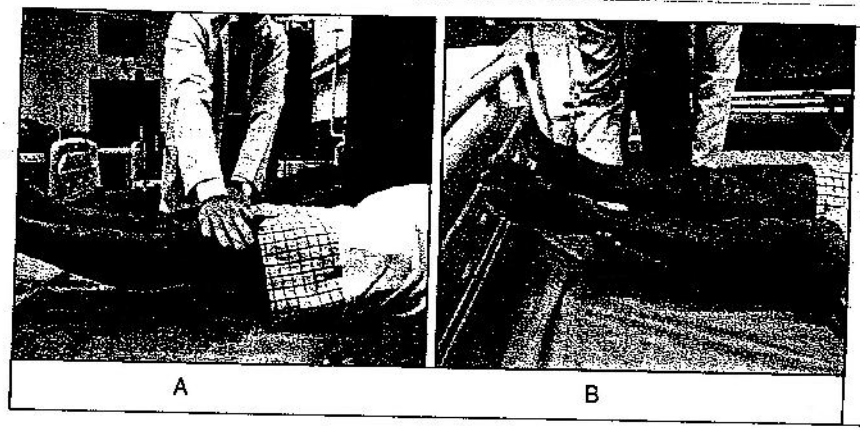
Ask the patient to lift up the straight leg and not let you push it down (having placed your hand above the knee).

THE MUSCLE(S)	NERVE ROOT(S)
Psoas and iliacus muscles	(L2, L3)

II. Test hip extension

Ask the patient to keep the leg down and not let you pull it up from underneath the calf or ankle.

THE MUSCLE(S)	NERVE ROOT(S)
Gluteus maximus	(L5, S1, S2)



A B

FIGURE 7 – 42. Clinical examination of hip flexion (A) and extension (B).

III. Test hip abduction

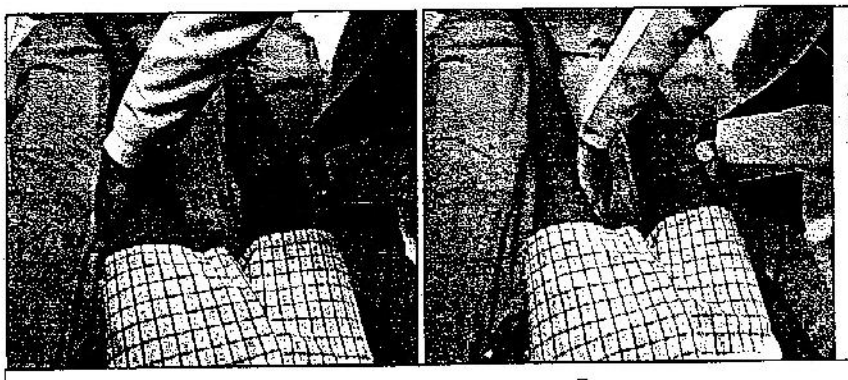
Ask the patient to abduct the leg and not let you push it in.

THE MUSCLE(S)	NERVE ROOT(S)
Gluteus medius and minimus, sartorius and tensor fasciae latae	(L4, L5, S1)

IV. Test hip adduction

Ask the patient to keep the leg adducted and not let you push it out.

THE MUSCLE(S)	NERVE ROOT(S)
Adductors longus, brevis and magnus	(L2, L3, L4)



A B

FIGURE 7 – 43. Clinical examination of hip abduction (A) and adduction (B).

THE KNEE (FIGURE 7 – 44)

I. Test Knee flexion

Ask the patient to bend the knee and not let you straighten it

THE MUSCLE(S)	NERVE ROOT(S)
Hamstrings (biceps femoris, semimembranosus, semitendinosus)	(L5, S1)

II. Test knee extension

With the knee slightly bent, ask the patient to straighten the knee and not let you bend it

THE MUSCLE(S)	NERVE ROOT(S)
Quadriceps femoris (this muscle is three times as strong as its antagonists, the hamstrings)	(L3, L4)

A B

FIGURE 7 – 44. Clinical examination of knee flexion (A) and extension (B).

THE ANKLE JOINT (FIGURE 7 – 45)

I. Test plantar flexion

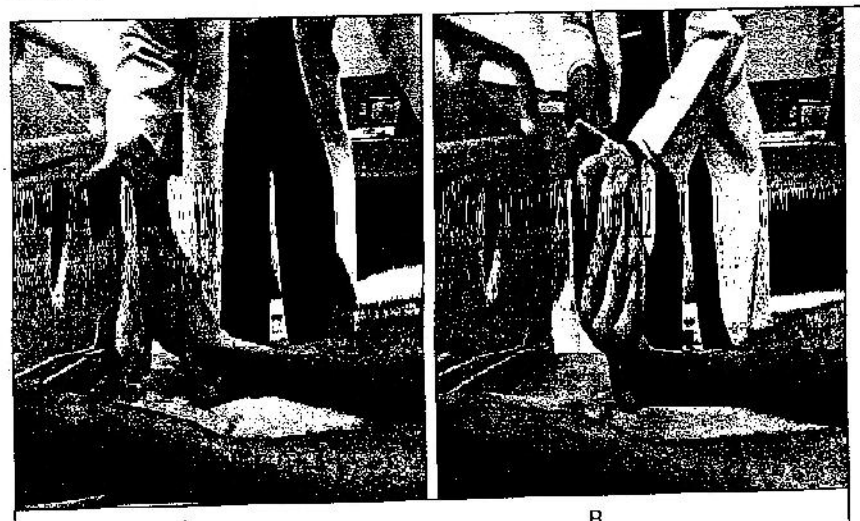
Ask the patient to push the foot down and not let you push it up

THE MUSCLE(S)	NERVE ROOT(S)
Gastrocnemius, plantaris, soleus	(S1, S2)

II. Test dorsiflexion

Ask the patient to bring the foot up and not let you push it down

THE MUSCLE(S)	NERVE ROOT(S)
Tibialis anterior, extensor digitorum longus and extensor hallucis longus	(L4, L5)



A B

FIGURE 7 – 45. Clinical examination of ankle planter flexion (A) and dorsiflexion (B).

THE TARSAL JOINT (FIGURE 7 – 46)

I. Test eversion

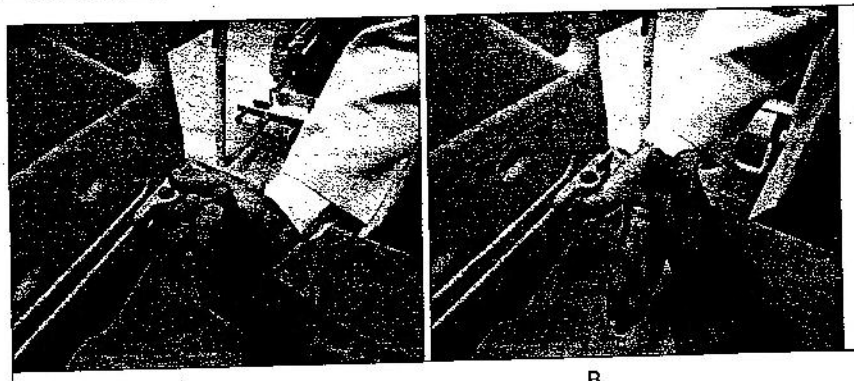
Ask the patient to evert the foot against resistance

THE MUSCLE(S)	NERVE ROOT(S)
Peroneus longus and brevis, and extensor digitorum longus	(L5, S1)

II. Test inversion

With the foot in complete plantar flexion, ask the patient to invert the foot against resistance

THE MUSCLE(S)	NERVE ROOT(S)
Tibialis posterior, gastrocnemius and hallucis longus	(L5, S1)



A B

FIGURE 7 – 46. Clinical examination of tarsal joint eversion (A) and inversion (B).

REFLEXES

Reflexes are automatic reactions to stimuli that occur without conscious thought (involuntarily) even though they involve skeletal muscles. Three types of reflexes are commonly examined. These are:

1 Superficial reflexes
2 Deep tendon reflexes
3 Primitive reflexes (frontal release signs)

SUPERFICIAL REFLEXES

The most commonly tested superficial reflexes are the abdominal, the cremasteric, and the planter reflex.

I. Abdominal reflex (FIGURE 7 – 47)

The abdominal superficial reflex (T8-T12) is elicited by having the patient lie on his or her back. An applicator stick or tongue blade is quickly stroked horizontally laterally to medially toward the umbilicus. The result is a contraction of the abdominal muscles, with the umbilicus deviating toward the stimulus. This reflex is lost in upper motor lesions (ipsilateral suprasegmental or contralateral pyramidal tract lesions) but will also be affected by lower motor neurone damage affecting T8-T12. They may be difficult to elicit in the obese, the elderly or those who have had abdominal surgery. The reflex is easily fatigued so that the most accurate assessment will be the first time that it is elicited. Rarely, an inverted response occurs in which stimulation on the abnormal side (where the reflexes are absent) produces contractions of the normal side (Beevor's sign).

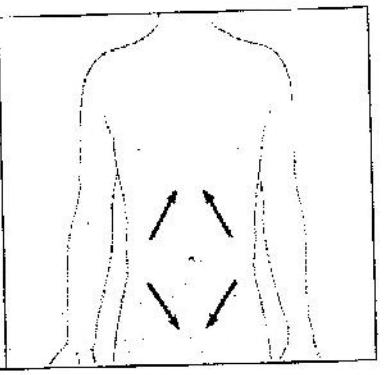


FIGURE 7 – 47. The superficial abdominal reflex is performed by strocking the abdominal wall with tongue blade.

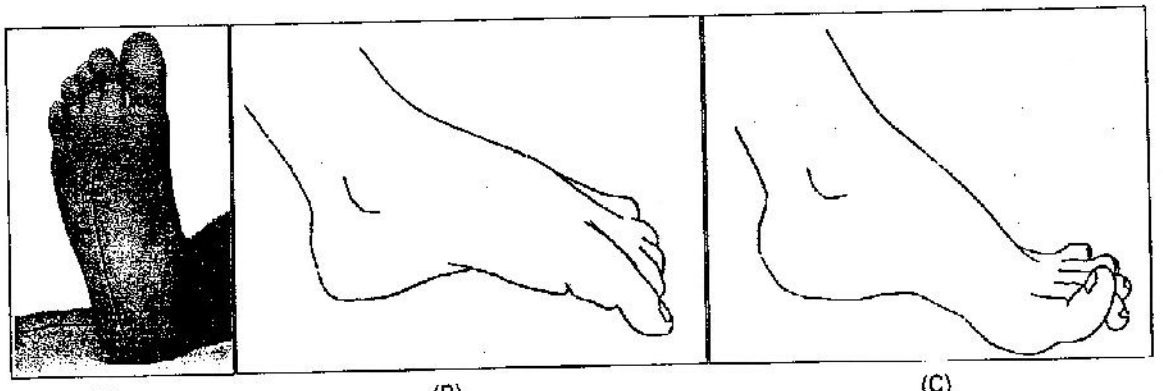
II. Cremasteric reflex

The cremasteric superficial reflex (L1 and L2) in men is elicited by lightly stroking the inner aspect of the thigh with an applicator stick or tongue depressor. The result is a rapid elevation of the testicle on the same side. The cremasteric reflex is used to assist in identifying the level of spinal cord lesions, particularly after injury.

III. Babinski's sign (planter reflex)

Babinski's sign or reflex is a pathologic reflex. The reflex tests nerve roots S1-S2. The technique of examination involves the following steps:

1 Warn the patient that you are about to touch the sole of their foot (this may prevent a startled withdrawal response).
2 Stroke the patient's sole with a key from the heel, up the lateral aspect of the sole to the base of the 5th toe. If there is no response, the stroke can be continued along the ball of the foot to the base of the big toe. This is shown in FIGURE 7 – 48
3 The normal response is flexion of the great toe and flexion of the other toes too (FIGURE 7 – 48).



(A) (B) (C)

FIGURE 7 – 48. The technique of planter reflex (A), the normal response (flexion response) (B), and the abnormal dorsiflexion (Babinski's sign) response (C).

CLINICAL
CLUE

FOR POSTGRADUATE STUDENTS

Pseudo-Babinski sign is the name given to dystonic extension of the great toe on stroking the sole of the foot, as when trying to elicit Babinski's sign, with which this may be confused, although pseudo-Babinski responses persist for longer, and spontaneous extension of the toe, striatal toe, may also be present. Pseudo- Babinski signs may normalize after dopaminergic treatment in dopa-responsive dystonia.

DEEP TENDON REFLEXES

A tendon reflex is mediated by a reflex arc consisting of an afferent (sensory) and an efferent (motor) neurone with one synapse between (a monosynaptic reflex). Muscle stretch activates the muscle spindles, which send a burst of afferent signals that in turn lead to direct efferent impulses, causing muscle contraction. These stretch reflex arcs are served by a particular spinal cord segment which is modified by the influence of descending upper motor neurones. The technique of deep tendon reflex (FIGURE 7 – 50) includes:

1. Support the joint being tested so that the muscle is relaxed
2. Palpate the tendon before striking with hammer
3. The reflex hammer is swung by motion at the wrist, not the elbow. Allow the weight of the reflex hammer head to determine the strength of the blow.
4. Gentle taps over the tendon being tested should produce muscle contraction. Two strikes are needed for each tendon jerk; in the first one look at the muscle contraction and in the second one look at the joint movement
5. Test each reflex, and compare it with the other side. Reflexes should be symmetrically equal. There is individual variation in the reflex response and several general points can be made regarding reflex asymmetries:
   - **A.** Reflexes that are brisker on one side of the body than on the other—usually indicate an upper motor neuron disturbance, but sometimes reflect a lower motor neuron lesion on the side with the depressed reflexes.
   - **B.** Focal reflex deficits often relate to root, plexus, or peripheral nerve lesions. For example, unilateral depression of the ankle jerk commonly reflects an S1 radiculopathy resulting from a lumbosacral disk lesion.
   - **C.** Loss of distal tendon reflexes (especially ankle jerks), with preservation of more proximal ones, is common in polyneuropathies.
6. If no response is obtained perform reinforcement (Jendrassik's) maneuver. This maneuver increases anterior horn cell excitability. If you use reinforcement, the patient must relax between repeated attempts. This effect is short-lived, however, so you should aim to test the reflex in the first 10 seconds of the reinforcement.
7. If the lower limb reflexes are so strong perform clonus maneuver.
8. Reflexes are commonly graded on a scale from 0 to 3 pluses (BOX 7 – 15)

FIGURE 7 – 50. The technique of deep tendon reflex.

BOX 7 – 15. Grading of deep reflexes

+++	Very brisk, hyperactive, with clonus (rhythmic oscillations between flexion and extension)
++	Average; normal
+	Somewhat diminished; low normal
±	When only present using reinforcement
0	No response

COMMENT ON PLANTER REFLEX AS FOLLOWS

1	Planter flexion response (normal response)	Flexion of the great toe and flexion of the other toes too
2	Dorsiflexion response (positive Babinski's sign)	Dorsiflexion of the big toe, with fanning of the other toes as shown in FIGURE 7 – 48
3	Intermediate response	Toes that seem to oscillate between flexion and extension are called "intermediate" and signify corticospinal disease
4	Withdrawal response and the test should be repeated	Leg is withdrawn and the heel moves in a ticklish reaction

If the etiology of the corticospinal dysfunction is in the spinal cord, Babinski signs are elicited prior to the hammer or key reaching the forefoot. The easier it is to elicit the response the more likely the lesion is at a spinal level. In patients with longstanding disease or those with severe lesions, the onset of the stimulus at S1 provokes dorsiflexion of the foot in association with hip and knee flexion with contraction of the tensor facia lata (a mass reflex). If the foot is amputated, a partial reflex (contraction of the tensor fascia lata) can often be attained from stimulating the stump. Causes of bilateral upgoing toes may be found in:

1 Upper motor neurone lesions (pyramidal lesion)
2 Infants up 18 months
3 Hypoglycemia
4 Deep sleep
5 Apnea
6 After generalised seizure
7 Comatose patient

Because Babinski's sign is an abnormal reflex, one should comment only that Babinski's sign is present; it is never absent. An extensor planter response can also be elicited, although less reliably, by such maneuvers as pricking the dorsal surface of the big toe with a pin (Bing sign), firmly stroking down the anterior border of the tibia from knee to ankle (Oppenheim maneuver), squeezing the calf muscle (Gordon maneuver) or Achilles tendon (Schafer maneuver), flicking the little toe (Gonda maneuver), or stroking the back of the foot just below the lateral malleolus (Chaddock maneuver). In interpreting responses, attention must be focused only on the direction in which the big toe first moves. These techniques are shown in FIGURE 7 – 49.

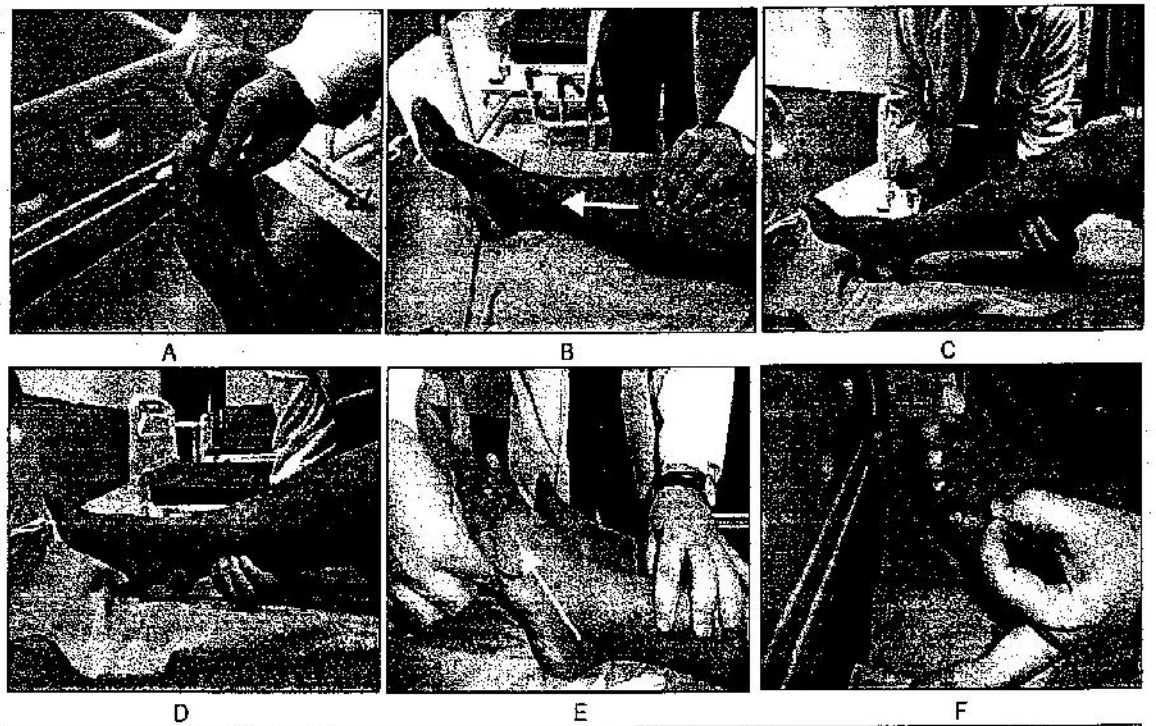


FIGURE 7 – 49. To elicit planter flexion response use Babinski's derivatives which include Bing sign (A), Oppenheim maneuver (B), Gordon maneuver (C), Schafer maneuver (D), Chaddock maneuver (E), and Gonda maneuver (F).

A few clonic beats (< 3 beats), termed non-sustained clonus, may be seen when the patient is tense or has exercised, hyperthyroidism, electrolyte abnormalities, and other metabolic abnormalities. Sustained clonus (> 3 clonic beats) is a sign of upper motor neuron lesion.

Patients with hypothyroidism have decreased relaxation phase of deep tendon reflex, which is termed a hung-up reflex. The maneuver is shown in FIGURE 7 – 52. In addition to hypothyroidism, delayed relaxation phase occurs in diabetes mellitus, syphilis, hypothermia, post-partum, anorexia nervosa, and treatment with β-blockers.

DEEP TENDON FEFLEXES OF THE UPPER LIMBS (FIGURE 7 – 53)

The sudden stretching of a muscle usually evokes brisk contraction of that muscle or muscle group. As the reflex is a response to stretching of a muscle it is correctly called a muscle stretch reflex rather than a tendon reflex. The tendon merely transmits stretch to the muscle. In Iraq, the Queen Square hammer is in common use.

I. Triceps jerk (C6, C7)

Flex the patient's arm at the elbow, with the palm toward the body, and pull it slightly across the chest. Strike the triceps tendon above the elbow. Watch for contraction of the triceps muscle and extension at the elbow.

Alternatively the triceps reflex can be elicited by hanging the patient's arm over your arm, as shown in FIGURE 7 – 53. Tapping the triceps tendon in this position often elicits the reflex.

II. Biceps jerk (C5, C6)

The patient's arm should be partially flexed at the elbow with palm down. Place your thumb or finger firmly on the biceps tendon. Strike with the reflex hammer so that the blow is aimed directly through your digit toward the biceps tendon. Observe flexion at the elbow, and watch the contraction of the biceps muscle.

III. Brachioradialis (supinator) jerk (C5, C6),

The patient's hand should rest on the abdomen or the lap, with the forearm partly pronated. Strike the lower end of the radius just above the wrist. Watch for flexion and supination of the forearm. To avoid hurting the patient by striking the radial nerve directly, place your own first two fingers over this spot and then strike the fingers, as with the biceps jerk.

IV. Finger jerk (C8)

Place your left hand below and across the palmer surface of the relaxed fingers of the patient. Then tap your fingers with the patellar hammer. Normally, slight flexion of all the fingers occurs.

V. Hoffmann reflex

With the patient's hand is pronated, the candidate grasps the terminal phalanx of the middle finger between the index finger and thumb. With a sharp jerk, the phalanx is passively flexed and suddenly released. A positive response consists of adduction and flexion of the thumb as well as flexion of the other fingers.

FIGURE 7 – 53. The technique of deep tendon reflexes including triceps jerk (A and B), biceps jerk (C), brachioradialis (D), finger jerk (E), and Hoffman jerk (F).

CLINICAL CLUE

If elbow extension and finger flexion is the only response when the patient's wrist is tapped during supinator jerk, the response is said to be inverted and is known as the inverted brachioradialis (supinator) jerk. This is associated with an absent biceps jerk and an exaggerated triceps jerk. It indicates a spinal cord lesion at the C5 or C6 level due, for example, to compression (e.g. disc prolapse), trauma or syringomyelia. It occurs because a lower motor neurone lesion at C5 or C6 is combined with an upper motor neurone lesion affecting the reflexes below this level

DEEP TENDON REFLEXES OF THE LOWER LIMBS

Deep tendon reflexes of lower limbs (FIGURE 7 – 54)

I. Knee jerk (L3, L4)

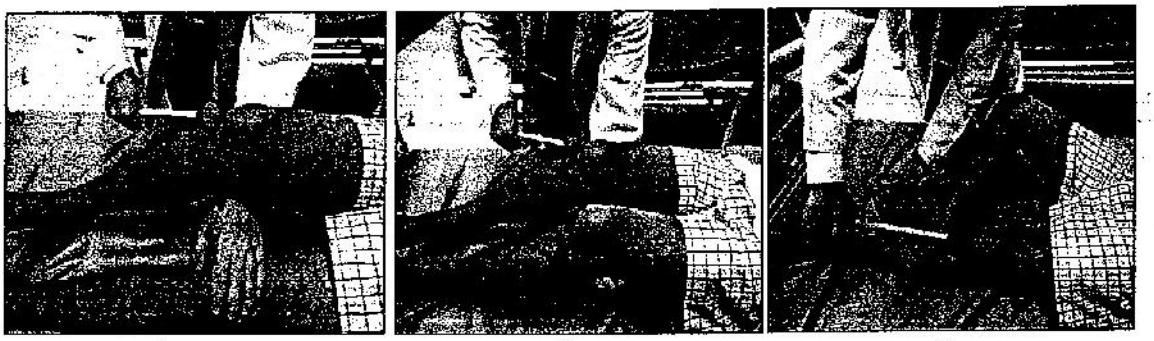
Two methods can be used to elicit knee jerk in supine patient. Firstly expose the patient's thigh and then:

Method I — Slide your left arm under the patient's right knee, raising it slightly and rest your arm over the patient's left knee. Then briskly tap the infrapatellar tendon (just below the patella). Note contraction of the quadriceps with extension at the knee.

Method II — Slide one arm under the knees so that they are slightly bent and supported. The tendon hammer is allowed to fall onto the infrapatellar tendon. Normally, contraction of the quadriceps causes extension of the knee

II. Ankle jerk (S1)

Have the foot in the mid-position at the ankle (90°) with the knee bent (90°), the thigh externally rotated on the bed, and the foot held in dorsiflexion by the candidate. The hammer is allowed to fall on the Achilles tendon. The normal response is plantar flexion of the foot with contraction of the gastrocnemius muscle.



A B C

FIGURE 7 – 54. The technique of deep tendon reflexes in the lower limbs including the knees (A and B) and the ankle jerk (C).

SPECIAL SITUATION

Sometimes, the knee jerk or ankle jerk is absent but planter response is extensor. In this case, one should think with one of the possibilities listed in BOX 7 – 16.

BOX 7 – 16. Causes of absent knee/ankle jerk and extensor planter response

Spinal causes	Non-spinal causes
Subacute combined degeneration of the spinal cord	Motor neuron disease
Spinal cord compression at the fourth lumber level	Friedrich's ataxia
Spinal shock	Diabetic amyotrophy
Conus medullaris	Meningeal carcinomatosis
Co-existing lumber and cervical spondylosis	Peripheral neuropathy in a stroke patient

PRIMITIVE REFLEXES (FRONTAL RELEASE SIGNS) (NON-ROUTINE SIGNS)

Frontal release signs are primitive reflexes that can be thought of as adaptive for the infants, such as grasping or sucking, but disappear as the brain matures. Diffuse neuronal dysfunction, especially frontal lobe damage, releases them from inhibition. These reflexes (FIGURE 7 – 55) include:

I. GRASP SIGN

The grasp response is elicited by lightly drawing handle of the hammer along the hypothenar (medial) aspect of the patient's palm and then transversely across the fingertips, gently lifting. This test is performed while the patient's attention is distracted. In a pronounced grasp response, the patient's fingers will hook around the candidate's fingers firmly. Even the touch of an inanimate object such as a chair arm may elicit a tenacious grasp.

II. SUCK SIGN (ROOTING REFLEX)

In this reflex, touching the lip or corner of the mouth produces head deviation to the side of the stimulus in association with a sucking movement of the lips.The same responses may occur at the mere sight of an approaching finger.

III. SNOUT SIGN

With an index finger positioned vertically over the lips, tapping the finger with the other hand or with a reflex hammer normally elicits protrusion of the lips an abnormal response is exaggerated protrusion.

IV. GLABELLAR SIGN (MYERSON SIGN)

This sign is elicited by repetitive tapping between the patient's eyebrows. The normal response is to blink a few times and then stop (the blink response normally stops after 3 or 4 taps). The response is abnormal if the patient continues to blink with each tap as long as the stimulus continues. The glabellar sign may also be present in extrapyramidal disorders.

V. PALMOMENTAL REFLEX

The edge of a reflex hammer is lightly stroked from medial to lateral across the thenar eminence. If the palmomental reflex is present, there will be a contraction of the ipsilateral mentalis seen in the neck and chin.

FIGURE 7 – 55. The technique of primitive reflexes including grasp reflex (A), suck sign (B), snout sign (C), glabellar sign (D), and palmomental reflex (E).

COORDINATION (CEREBELAR SIGNS)

The cerebellum has multiple connections (afferent and efferent) to sensory pathways, brainstem nuclei, the thalamus and the cerebral cortex. Via these connections the cerebellum plays an integral role in coordinating voluntary movement. Clinical signs of cerebellar lesion are shown in BOX 7 – 17. A standard series of simple tests is used to test coordination. Always demonstrate these movements for the patient's benefit.

BOX 7 – 17. Clinical signs of cerebellar lesion

Nystagmus	Hypotonia
Speech disorder (dysarthria)	Cerebellar (truncal) ataxia
Pronator drift	Pendular knee jerk
Intension tremor	Impaired ability to generate alternating rhythmic movements (dysdiadochokinesis)
Past pointing (overshooting or dysmetria)	Rebound phenomenon
Dyssynergia	

A lesion in a cerebellar hemisphere causes lack of coordination, and the clinical signs listed in BOX 7 – 17 on the same side of the body. In disorders which predominantly affect midline cerebellar structures, e.g. tumours of the vermis and alcoholic cerebellar damage, check the gait, as truncat ataxia may be the only abnormal finding.

UPPER LIMBS CEREBELLAR TESTS (FIGURE 7 – 56)

I. The finger nose test

Ask the patient to touch your index finger and then his/her nose alternately several times. Move your finger about so that the patient has to alter directions and extend the arm fully to reach it. Repeat the test on the contralateral hand and observe the accuracy and smoothness of movements and watch for any tremor. Normally the patient's movements are smooth and accurate. In cerebellar lesion, the finger-nose test may reveal a tendency to fall short or overshoot the candidate's finger (past-pointing or overshooting or dysmetria). In more severe cases there may be a tremor of the finger as it approaches the target finger and the patient's own nose (intention tremor). The movement may be clumsy, unsteady, and varying in their speed and direction (dyssynergia).

II. Rebound phenomenon

With the patient sitting and out stretch both hands, stand behind and continuously press the arm. Patient with cerebellar incoordination, the sudden withdrawal of both hands resulting in high degree of the elevation of the arms or even the patient may fall backwards.

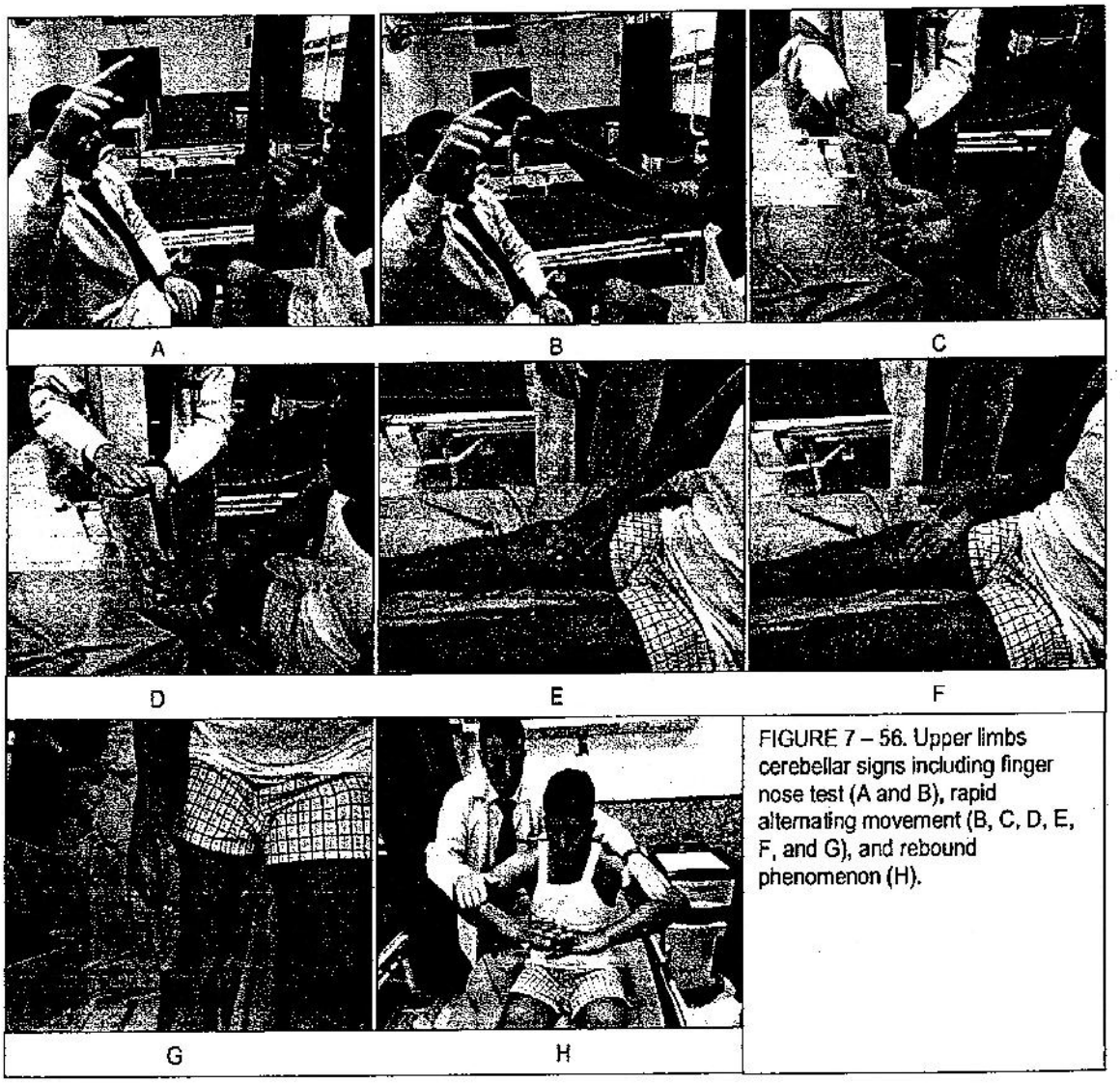


FIGURE 7 – 56. Upper limbs cerebellar signs including finger nose test (A and B), rapid alternating movement (B, C, D, E, F, and G), and rebound phenomenon (H).

III. Rapid alternating movements

The ability to perform rapid alternating movements is called diadochokinesia. The patient can be asked to pronate and supinate one hand on the other hand rapidly. Another technique involves having the patient touch the thumb to each finger as quickly as possible. The patient may also be asked to slap the thigh, raise the hand, turn it over, and slap the

thigh again rapidly. This pattern is repeated on the other hand. An abnormality in performing rapid alternating movements is called dysdiadochokinesis.

LOWER LIMB CEREBELLAR SIGNS (FIGURE 7 – 57)

I. Heel-shin test

Ask the patient to slide the heel of one foot along shin of the opposite leg at a moderate pace and as accurately as possible. Look for past pointing, dyssynergia, and intention tremor.

II. Pendular knee jerk

Cerebellar lesions produce a pendular oscillating reflex that may take more than five oscillations before it stops. It has large amplitude because antagonist muscles are not engaged in the normal fashion. Pendular jerks are best seen in the knee. While the patient is sitting, ask him/her to put the legs over the side of the bed and test for pendular knee jerks.

FIGURE 7 – 57. Upper limbs cerebellar signs including finger nose test (A), rapid alternating movement (B and C), and rebound phenomenon (D).

CLINICAL CLUE

If the order of examination is perform cerebellar tests (upper limbs or lower limbs), the candidate should take permission from the examination committee to perform the following related tests:

1. Speech (dysarthria)
2. H – test (nystagmus)
3. Tone examination (hypotonia)
4. Pronator drift examination (cerebellar drift)
5. Stance and gait (cerebellar/truncal ataxia)

Pronator drift and stance and gait examination will be discussed later.

INTERPRETATION OF THE MOTOR FINDINGS

The descending pathway (FIGURE 7 – 58) is involved in the control of the motor function and consists of two primary groups of neurons. These are:

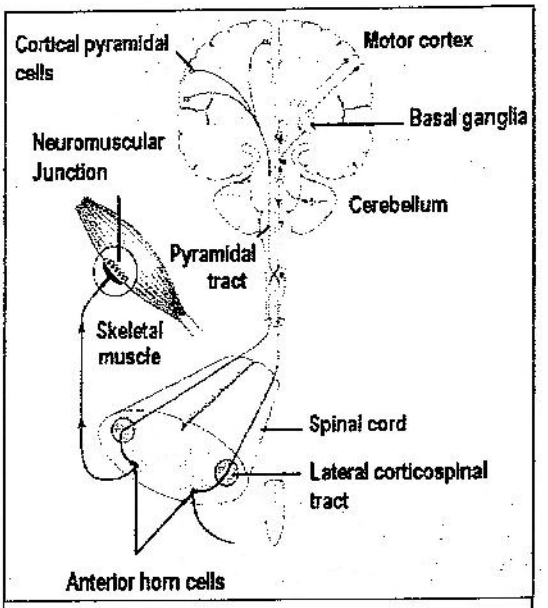


FIGURE 7 – 58. The motor system.

UPPER MOTOR NEURONS

These neurons originate from the cerebral cortex, cerebellum and brain stem (except nuclei of cranial nerves) and modulate the activity of the lower motor neurons. Their fibers comprise the descending motor pathway which is subdivided into two groups

1 The pyramidal system (their fibers pass through the pyramid of the medulla oblongata) involves in conscious motor function. Neurons from the motor cortex descend as the pyramidal tract in the internal capsule and the ventral brain stem before crossing in the medulla to enter the lateral columns of the spinal cord. In the spinal cord, the pyramidal tract (upper motor neurons) form the corticospinal tract in the lateral column and end by synapsing with the anterior horn cells of the spinal cord grey matter, which form the lower motor neurons

2 The extrapyramidal system (their fibers do not pass through pyramid of the medulla oblongata) involves in the unconscious motor function

LOWER MOTOR NEURONS

These neurons present either in the anterior horn of the central gray matter of the spinal cord or in the cranial nerve nuclei of the brain stem. The axons of the lower motor neuron extend through peripheral nerves to the skeletal muscles

Any lesion affects the upper motor neurons or their axons in cerebral cortex, subcortical white matter, internal capsule, brain stem (except nuclei of cranial nerves) and the nerve tracts up to the lower motor neuron produces upper motor neuron lesion signs while any lesion affects anterior horn cells of the spinal cord, motor root and motor nerve produces lower motor neuron lesion signs. These are contrasted in BOX 7 – 18.

BOX 7 – 18. Physical signs in different types of motor deficit

Clinical sign	Upper motor (pyramidal) lesion	Lower motor lesion	Extrapyramidal lesion	Cerebellar lesion
Power	Weak Upper limbs: extensors weaker Lower limbs: flexors weaker	Weak	No weakness	No weakness
Wasting	None	Yes, after interval	None	None
Fasciculation	None	Yes, after interval	None	None
Tone	Spastic increase (after interval)	Flaccid from onset	Rigidity (cogwheel)	Normal/ reduced
Reflexes	Increased	Reduced/absent	Normal	Normal
Plantar response	Extensor	Flexor	Flexor	Flexor
Coordination	Reduced by weakness	Reduced by weakness	Normal (but slowed)	Impaired

Three kinds of motor pathways impinge on the anterior horn cells of the spinal cord: the corticospinal tract, the basal ganglia system, and the cerebellar system. All of these higher motor pathways affect movement only through the lower motor neurons.

When the corticospinal tract is damaged or destroyed, its functions are reduced or lost below the level of injury. Monoplegia denotes paralysis or severe weakness of the muscles in one limb, and monoparesis denotes less severe weakness in one limb, although the two words are often used interchangeably. Hemiplegia or hemiparesis is weakness in both limbs (and sometimes the face) on one side of the body; paraplegia or paraperesis is weakness of both legs; and quadriplegia or quadriparesis (also tetraplegia, tetraparesis) is weakens of all four limbs.

When upper motor neurons are damaged above the crossover of its tracts in the medulla, motor impairment develops on the opposite or contralateral side. In damage below the crossover, motor impairment occurs on the same or ipsilateral side of the body. Diseases of the basal ganglia system or cerebellar system do not cause paralysis, but can be disabling.

UPPER MOTOR NEURONE LESIONS

In general, distal muscle groups are affected more severely than proximal ones, and axial movements are spared unless the lesion is severe and bilateral. With corticobulbar (tract of cranial nerves) involvement, weakness is usually observed only in the lower face and tongue; extraocular, upper facial, pharyngeal, and jaw muscles are almost always spared. With bilateral corticobulbar lesions, pseudobulbar palsy often develops. With the involvement of the upper motor neurones, the following signs occur but further clinical findings depend upon the actual site of the lesion.

1 Weakness or paralysis
2 Little, if any, muscle atrophy
3 Hypertonia (spasticity)
4 Loss of superficial abdominal reflexes
5 An extensor plantar (Babinski) response
6 Increased tendon reflexes

Localization of the underlying lesion

1. A parasagittal intracranial lesion produces an upper motor neuron deficit that characteristically affects both legs (paraperesis) and may later involve the arms. Causes of paraperesis are shown in BOX 7 – 19.

BOX 7 – 19. Causes of paraperesis (paraplegia)	
Demyelination e.g. multiple sclerosis	Syringomyelia
Cord compression e.g. trauma and tumor	Hereditary spastic paraperesis
Parasagittal lesion e.g. meningioma	Osteoarthritis of the cervical cord
Infection e.g. HTLV and HIV	

2. A discrete lesion of the cerebral cortex or its projections may produce a focal motor deficit involving, for example, the contralateral hand. Weakness may be restricted to the contralateral leg in patients with anterior cerebral artery occlusion or to the contralateral face and arm if the middle cerebral artery is involved. A more extensive cortical or subcortical lesion will produce weakness or paralysis of the contralateral face, arm, and leg and may be accompanied by aphasia, a visual field defect, or a sensory disturbance of cortical type.
3. A lesion at the level of the internal capsule, where the descending fibers from the cerebral cortex are closely packed, commonly results in a severe hemiparesis that involves the contralateral limbs and face.
4. A brainstem lesion commonly—but not invariably—leads to bilateral motor deficits, often with accompanying sensory and cranial nerve disturbances, and disequilibrium. A more limited lesion involving the brainstem characteristically leads to a cranial nerve disturbance on the ipsilateral side and a contralateral hemiparesis (crossed hemiplegia); the cranial nerves affected depend on the level at which the brainstem is involved.
5. A spinal cord lesion is discussed later.

LOWER MOTOR NEURONE LESION

This pattern results from disorders of cell bodies of lower motor neurons in the brainstem motor nuclei and the anterior horn of the spinal cord, or from dysfunction of the axons of these neurons as they pass to skeletal muscle.
The following signs occur with involvement of the lower motor neuron:

1 Weakness or paralysis
2 Wasting and fasciculations of involved muscles
3 Hypotonia (flaccidity)
4 Normal abdominal and plantar reflexes—unless the neurons subserving them are directly involved, in which case reflex responses are lost
5 Loss of tendon reflexes when neurons subserving them are affected

Localization of the underlying lesion

In distinguishing weakness from a root, plexus, or peripheral nerve lesion (discussed later), the distribution of the motor deficit is of particular importance. Only those muscles supplied wholly or partly by the involved structure are weak. The distribution of any accompanying sensory deficit similarly reflects the location of the underlying lesion.

EXTRAPYRAMIDAL LESION

Lesions of the extrapyramidal system often produce:

1 No weakness
2 No muscle atrophy

3 Hypertonia (rigidity)
4 Normal superficial abdominal reflexes
5 Normal planter response
6 Normal tendon reflexes
7 Bradykinesia (rapid movements are slowed and clumsy)
8 Extrapyramidal lesions also cause postural instability, monotonous speech, and resting tremor,

HOW TO EXAMINE FOR BRADYKINESIA (FOR POSTGRADUATE STUDENTS)
Two simple tests (FIGURE 7 – 59) for bradykinesia are finger tapping and twiddling. Ask the patient to tap the fingers in turn onto a surface repeatedly, quickly and with both hands at once. Twiddling is rotating the hands around each other in front of the body. These movements are slow and clumsy in Parkinsonian patients but obviously depend on motor and cerebellar function as well. Difficulty getting out of a chair can be another sign of bradykinesia and patients often have difficulty turning over in bed.

A B

FIGURE 7 – 59. Tests for bradykinesia including finger tapping (A) and twiddling (B) tests.

CLINICAL CLUE

If the order of examination is perform relevant extrapyramidal system tests, the postgraduate candidate should perform the usual motor examination and then take permission from the examination committee to perform the following related tests:

6. Speech (dysarthria)
7. H – test (up ward gaze palsy)
8. Stance and gait (fastinant shuffling gait)
9. The glabellar tap

Stance and gait examination will be discussed later.

CEREBELLAR DYSFUNCTION
The physical signs of cerebellar dysfunction are discussed earlier.

MYOPATHIC DISORDERS
Myopathic weakness is produced by disorders of the muscle fibers. At a muscle fiber, if the nerve terminal releases a normal number of acetylcholine molecules presynaptically and a sufficient number of postsynaptic acetylcholine receptors are opened, the end plate reaches threshold and thereby generates an action potential that spreads across the muscle fiber membrane and into the transverse tubular system. Myopathic weakness is produced by a decrease in the number or contractile force of muscle fibers activated within motor units e.g. muscular dystrophies and inflammatory myopathies. The following signs usually occur:

1 Weakness, usually most marked proximally rather than distally
2 No muscle wasting until at least an advanced stage of the disorder
3 Hypotonia (muscle diseases)
4 Normal superficial abdominal and planter response reflexes until at least an advanced stage of the disorder
5 Normal tendon reflexes until at least an advanced stage of the disorder
6 No sensory loss or sphincter disturbances

NEUROMUSCULAR TRANSMISSION DISORDERS

Diseases of the neuromuscular junction, such as myasthenia gravis, produce weakness in a similar manner to myopathic lesion, but the loss of muscle fibers is functional (due to inability to activate them) rather than related to muscle fiber loss. Thus, fatigable weakness is suggestive of myasthenia gravis or other disorders of the neuromuscular junction. The following signs usually occur:

1 Fatigable weakness, often patchy, not conforming to the distribution of any single anatomic structure; frequently involves the cranial muscles and may fluctuate in severity over short periods, particularly in relation to activity.
2 No muscle wasting
3 Fatigable muscle tone
4 Normal or reduced (fatigable) superficial abdominal and planter response reflexes
5 Normal or reduced (fatigable) tendon reflexes
5 No sensory loss or sphincter disturbances

HOW TO EXAMINE FOR FATIGUABLE MUSCLE WEAKNESS (FOR POSTGRADUATE STUDENTS)

It is necessary in patient suspected of having myasthenia gravis to test for muscle fatigue (FIGURE 7 – 60). Three tests are generally designed for muscle weakness.

1 Test the oculomotor muscles by asking the patient to sustain upward gaze by looking up at the ceiling for one minute, and watch for progressive ptosis
2 Test the Peek sign for orbicularis oculi weakness. Ask the patient to close the eyes; if positive, within 30 seconds the lid margin will begin to separate, showing the sclera. This test strongly increases the likelihood of myasthenia.
3 Then test the proximal limb girdle muscles by asking the patient to hold the arms above the head. The examiner can repeatedly press the abducted arms down until they weaken. Power will decrease with repeated muscle contraction

FIGURE 7 – 60. Tests for fatigable muscle weakness including ceiling test (A), Peek sign (B), and proximal muscle weakness test (C).

SPINAL CORD LESION

The individual vertebrae are separated by intervertebral disc that consists of a thick outer fibrous portion called the annulus fibrosus, within which is a soft, gelatinous, inner central portion called the nucleus pulposus. Posterior to the vertebral bodies, the vertebral arches (composed of paired pedicles anteriorly and laminae posteriorly) and transverse processes enclose the spinal cord in the spinal canal, and the posterior spinous process projects posteriorly (FIGURE 7 – 61). Paraspinal muscles help support the spine. The spinal cord is a thin, tubular extension of the central nervous system that originates at the medulla and continues caudally to the conus

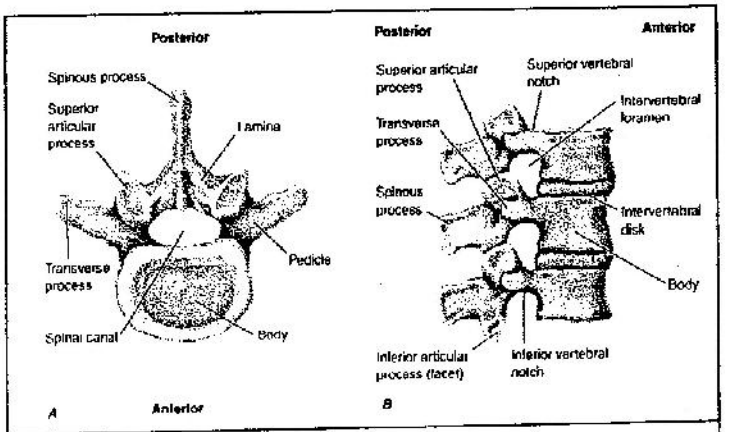


FIGURE 7 – 61. Anatomy of the vertebral column (A and B).

medullaris at the lumbar level; its fibrous extension, the filum terminale, terminates at the coccyx (FIGURE 7 – 3). The adult spinal cord is enlarged in the cervical and lumbar regions, where neurons that innervate the upper and lower extremities, respectively, are located. The white matter tracts containing ascending sensory and descending motor pathways are located peripherally, whereas nerve cell bodies are clustered in an inner region. The membranes that cover the spinal cord—the pia, arachnoid, and dura—are continuous with those of the brain. The spinal nerves are grouped into 8 cervical (C1-C8), 12 thoracic (T1-T12), 5 lumbar (L1-L5), 5 sacral (S1-S5), and 1 coccygeal nerve.
Note that the spinal cord is not as long as the vertebral canal and it ends at L1 – L 2. Because of this, the nerve roots exit at a level above their respective vertebral bodies in the cervical region (the C7 nerve root exits at the C6-C7 level) and below their respective vertebral bodies in the thoracic and lumbar regions (the T1 nerve root exits at the T1-T2 level). The cervical nerve roots follow a short intraspinal course before exiting. By contrast, because the spinal cord ends at the vertebral L1 or L2 level, the lumbar nerve roots follow a long intraspinal course and can be injured anywhere from the upper lumbar spine to their exit at the intervertebral foramen. For example, disk herniation at the L4-L5 vertebral level commonly produces compression of the traversing S1 nerve root.

RULE

With compressive and other focal lesions that involve the anterior horn cells in addition to the fiber tracts traversing the cord, the muscles innervated by the affected cord segment weaken and atrophy. Therefore, a focal LMN deficit exists at the level of the lesion and an UMN deficit exists below it—in addition to any associated sensory disturbance

APPRAOCH TO THE PATIENT

The presence of a horizontally defined level below which sensory, motor, and autonomic function is impaired is a hallmark of spinal cord disease. This sensory level is sought by asking the patient to identify a pinprick or cold stimulus and sequentially moved up toward the neck on each side. The sensory level indicates damage to the spinothalamic tract one to two segments above the perceived level of a unilateral spinal cord lesion and at the level of a bilateral lesion. Lesions that transect the descending corticospinal and other motor tracts cause paraplegia or quadriplegia, with the evolution over time of increased muscle tone, heightened deep tendon reflexes, and Babinski signs (the upper motor neuron syndrome). Such lesions also typically produce autonomic disturbances consisting of absent sweating below the implicated cord level and bladder, bowel, and sexual dysfunction.
The uppermost level of a spinal cord lesion can also be localized by attention to the segmental signs corresponding to disturbed motor or sensory innervation by an individual cord segment. A band of altered sensation (hyperalgesia or hyperpathia) at the upper end of the sensory disturbance, fasciculations or atrophy in muscles innervated by one or several segments, or a muted or absent deep tendon reflex may be noted at this level. These signs also occur with focal root or peripheral nerve disorders; thus, segmental signs are most useful when they occur together with signs of long tract damage. With severe and acute transverse lesions, the limbs initially may be flaccid rather than spastic. This state of "spinal shock" lasts for several days, rarely for weeks, and should not be mistaken for extensive damage to many segments of the cord or for an acute polyneuropathy.
The main features of transverse damage at each level of the spinal cord are:

1 Cervical Cord

Above C4 lesions produce quadriplegia and weakness of the diaphragm. Lesions at C4-C5 produce quadriplegia; at C5-C6, there is loss of power and reflexes in the biceps and brachioradialis; at C7 level weakness is found in finger and triceps; and at C8, there will be lower motor neurone weakness and wasting of the intrinsic muscles of the hand. Horner's syndrome (miosis, ptosis, and facial hypohidrosis) may accompany a cervical cord lesion at any level

2 Thoracic Cord

Lesions here are localized by the sensory level on the trunk and by the site of midline back pain if it accompanies syndrome. Useful markers for localization are nipples (T4) and umbilicus (T10). Leg weakness and disturbances of bladder and bowel function accompany paralysis. Beevor's sign here is of value.

3 Lumbar Cord

Lesions at the L2-L4 spinal cord levels abolish the patellar (knee) reflex. Lesions at L5-S1 abolish the ankle jerks (S1)

4 Sacral Cord/Conus Medullaris

The conus medullaris is the tapered caudal termination of the spinal cord, comprising the lower sacral and single coccygeal segments. The conus syndrome is distinctive, consisting of bilateral saddle anesthesia (S3-S5), prominent bladder and bowel dysfunction (urinary retention and incontinence with lax anal tone), and impotence. The bulbocavernosus (Squeezing glans penis or clitoris normally causes anal sphincter contraction; mediated by S2-S4) and anal wink (contraction of the anal sphincter when the perianal skin is scratched; mediated by S4-S5) reflexes are absent. Muscle strength is largely preserved

5 Cauda Equina Syndrome

A cauda equina syndrome results from pathological processes affecting the spinal roots below the termination of the spinal cord around L1/L2, hence it is a syndrome of multiple radiculopathies. Depending on precisely which roots are affected, this may produce symmetrical or asymmetrical sensory impairment in the buttocks (saddle anaesthesia;

sacral anaesthesia) and the backs of the thighs, radicular pain, and lower motor neurone type weakness of the foot and/or toes (even a flail foot). Weakness of hip flexion (L1) does not occur, and this together with relative sparing of bowel and bladder function may be useful in differentiating a cauda equina syndrome from conus medullaris lesion. Mass lesions in lower spinal canal often produce a mixed clinical picture in which elements of both cauda equina and conus medullaris coexist

6 Brown-Sequard Hemicord Syndrome

This consists of ipsilateral weakness (corticospinal tract) and loss of joint position and vibratory sense (posterior column), with contralateral loss of pain and temperature sense (spinothalamic tract) one or two levels below the lesion

7 Central Cord Syndrome

The central cord syndrome results from damage to the gray matter nerve cells and crossing spinothalamic tracts near the central canal. In the cervical cord, the central cord syndrome produces arm weakness out of proportion to leg weakness and a "dissociated" sensory loss, signifying a loss of pain and temperature sense in a cape distribution over the shoulders, lower neck, and upper trunk in contrast to preservation of joint position and vibration sense in these regions. Syringomyelia, tumors, and anterior spinal artery ischemia (including from aortic dissection) are the main causes

8 Anterior Spinal Artery Syndrome

This results from occlusion or diminished flow in artery. All spinal cord functions; motor, sensory, and autonomic—are lost bilaterally below level of the lesion, with the striking exception of retained vibration and position sensation

9 Foramen Magnum Syndrome

Lesions in this area interrupt decussating pyramidal tract fibers destined for the legs, which cross caudal to those of the arms, resulting in weakness of the legs (crural paresis). Compressive lesions near the foramen magnum may produce weakness of the ipsilateral shoulder and arm followed by weakness of the ipsilateral leg, then the contralateral leg, and finally the contralateral arm, an "around the clock" pattern that may begin in any of the four limbs. There is typically suboccipital pain spreading to the neck and shoulders

10 Intramedullary and Extramedullary Syndromes

It is useful to differentiate intramedullary processes, arising within the substance of the cord, from extramedullary ones that compress the spinal cord or its vascular supply. The differentiating features are only relative and serve as clinical guides. With extramedullary lesions, radicular pain is often prominent, and there is early sacral sensory loss (lateral spinothalamic tract) and spastic weakness in the legs (corticospinal tract) due to the superficial location of leg fibers in the corticospinal tract. Intramedullary lesions tend to produce poorly localized burning pain rather than radicular pain and spare sensation in the perineal and sacral areas ("sacral sparing"), reflecting the laminated configuration of the spinothalamic tract with sacral fibers outermost; corticospinal tract signs appear later. Regarding extramedullary lesions, a further distinction is made between extradural and intradural masses, as the former are generally malignant and the latter benign (neurofibroma being a common cause). Consequently, a long duration of symptoms favors an intradural origin

EXAMPLES

Examine the motor system of the upper limbs of this patient

Examination of the motor system had shown that the patient was lying in comfortable dorsal decubitus posture with no abnormal position, no involuntary movements, and normal muscle bulk; normal muscles tones of the upper limbs with no hypertonia, hypotonia, or paratonia; muscle power was full and grade five at the fingers, wrists, elbows, and shoulders bilaterally; two pluses (average or normal) triceps reflex, biceps reflex, supinator reflex, and finger reflex. No Hoffmann's reflex; good cerebellar coordination with no intension tremor, no overshooting, no dyssynergia, no dysdiadochokinesis, and no rebound phenomenon.

What is the interpretation?

The interpretation is normal motor system examination of the upper limbs.

Examination of the motor system of the upper limbs had shown that the patient was lying in dorsal decubitus posture with the left arm held tight the side. The fingers, wrist, and elbow were flexed. No involuntary movements can be noticed or elicited by flicking. Slight decrement in the muscle bulk of the left arm. The right arm shows normal position, normal muscle bulk, and no involuntary movements. Increased muscles tones of the left arm in the form of spasticity while the right arm

EXAMPLES

had normal muscle tone with no hypertonia, hypotonia, or paratonia. Muscle power of the left arm had shown that the extensor muscles were weak as compared with the flexor group (grade 0 vs grade 2); the right arm power was full and grade five at the fingers, wrist, elbow, and shoulder. The triceps, biceps, and supinator reflexes of the left arm were three pluses (hyperreflexia); two pluses (average or normal) triceps reflex, biceps reflex, supinator reflex, and finger reflex of the right arm. Hoffmann's sign can be elicited in the left arm. Cerebellar coordination tests can not be elicited in the left arm because of weakness; the right side had accepted cerebellar coordination with no intension tremor, no overshooting, no dyssynergia, no dysdiadochokinesis, and no rebound phenomenon.

What is the interpretation?
The interpretation is UMN lesion of the left arm e.g. old stroke.

Examination of the motor system of the upper limbs had shown that the patient was lying in dorsal decubitus posture with flaccid left arm. Fasciculations had been provoked by flicking the skin over the deltoid muscles. There was wasting of the muscles of the whole left upper arm. The right upper arm shows normal position, normal muscle bulk, and no involuntary movements. The muscle tone of the left arm was decreased (hypotonia, flaccid) while the right arm had normal muscle tone with no hypertonia, hypotonia, or paratonia. Grade 0 (very weak) muscle power of the left arm; the right arm power was full and grade five at the fingers, wrist, elbow, and shoulder. The triceps, biceps, and supinator reflexes of the left arm were absent even with reinforcement (grade 0); two pluses (average or normal) triceps reflex, biceps reflex, supinator reflex, and finger reflex of the right arm. Cerebellar coordination tests can not be elicited in the left arm because of weakness; the right side had accepted cerebellar coordination with no intension tremor, no overshooting, no dyssynergia, no dysdiadochokinesis, and no rebound phenomenon.

What is the interpretation?
The interpretation is LMN lesion of the left arm e.g. peripheral neuropathy.

Examination of the motor system of the upper limbs had shown that the patient was lying in dorsal decubitus posture with no wasting, weakness, or abnormal movements in both arms. The muscle tone of the left arm was slightly reduced (hypotonia) while the right arm had normal muscle tone with no hypertonia, hypotonia, or paratonia. The triceps, biceps, and supinator reflexes of both arms were normal with reinforcement (grade ±); no Hoffmann sign. Cerebellar coordination tests had shown the patient had intension tremor, overshooting, dyssynergia, dysdiadochokinesis, and rebound phenomenon of the left side; the right side had shown normal cerebellar function.

What is the interpretation?
The interpretation is left cerebellar hemisphere lesion.

Examination of the motor system of the upper limbs had shown that the patient was lying in dorsal decubitus posture with no wasting or weakness; resting tremor had been noticed in both arms. The muscle tone of both arms was increased (hypertonia) in the form of rigidity; cogwheel rigidity can be noticed in both wrists. The triceps, biceps, and supinator reflexes of both arms were normal (grade 2); no Hoffmann sign. The patient had slowed coordination in both arms.

What is the interpretation?
The interpretation is extrapyramidal lesion e.g. Parkinson's disease.

EXAMPLES

Examine the motor system of the lower limbs of this patient

Examination of the motor system of the lower limbs had shown that the patient was lying in comfortable dorsal decubitus posture with no abnormal positioning, no involuntary movements, and normal muscle bulk; normal muscles tones of the lower limbs with no hypertonia, hypotonia, or paratonia; muscle power was full and grade five at the hips, knees, ankles, and tarsal joints bilaterally; two pluses (average or normal) knee reflex and ankle reflex with flexor planter response. Good cerebellar coordination with no intension tremor, no overshooting, and no dyssynergia.

What is the interpretation?

The interpretation is normal motor system examination of the lower limbs.

Examination of the motor system of the lower limbs had shown that the patient was lying in dorsal decubitus posture with the left leg being extended and internally rotated. No involuntary movements can be noticed or elicited by flicking. Slight decrement in the muscle bulk of the left leg. The right leg shows normal position, normal muscle bulk, and no involuntary movements. Increased muscles tones of the left leg (hypertonia) in the form of spasticity while the right leg had normal muscle tone with no hypertonia, hypotonia, or paratonia. Muscle power of the left leg had shown that the flexor muscles were weak as compared with the extensor group (grade 0 vs grade 2); the right leg power was full and grade five at the hip, knee, ankle, and tarsal joint. The knee and the ankle reflexes of the left leg were three pluses (hyperreflexia) with sustained ankle clonus; two pluses (average or normal) knee and ankle reflexes of the right leg. Extensor planter response of the left leg and flexor planter response of the right leg. Cerebellar coordination tests can not be elicited in the left leg because of weakness; the right side had accepted cerebellar coordination with no intension tremor, no overshooting, and no dyssynergia.

What is the interpretation?

The interpretation is an UMN lesion of the left leg e.g. old stroke.

Examination of the motor system of the lower limbs had shown that the patient was lying in dorsal decubitus posture; no involuntary movements can be noticed or elicited by flicking; no decrement in the muscle bulk of the both legs. Increased muscles tones of the left leg (hypertonia) in the form of spasticity while the right leg had normal muscle tone with no hypertonia, hypotonia, or paratonia. Muscle power of the left leg had shown that the flexor muscles were weak as compared with the extensor group (grade 0 vs grade 2); the right leg power was full and grade five at the hip, knee, ankle, and tarsal joint. The knee and the ankle reflexes of the left leg were three pluses (hyperreflexia) with sustained ankle clonus; two pluses (average or normal) knee and ankle reflexes of the right leg. Extensor planter response of the left leg and flexor planter response of the right leg. Cerebellar coordination tests can not be elicited in the left leg because of weakness; the right side had accepted cerebellar coordination with no intension tremor, no overshooting, and no dyssynergia.

What is the interpretation?

The interpretation is an UMN lesion of the left leg e.g. established stroke.

Examination of the motor system of the lower limbs had shown that the patient was lying in dorsal decubitus posture with flaccid left leg. Fasciculations had been provoked by flicking the skin over the quadriceps muscles. There was wasting of the muscles of the whole left lower leg. The right leg showed normal position, normal muscle bulk, and no involuntary movements. The muscle tone of the left leg was decreased (hypotonia, flaccid) while the right leg had normal muscle tone with no hypertonia, hypotonia, or paratonia.

EXAMPLES

Grade 0 (very weak) muscle power of the left leg; the right leg power was full and grade five at the hip, knee, ankle, and tarsal joint. The knee and the ankle reflexes of the left leg were absent even with reinforcement (grade 0); two pluses (average or normal) knee and ankle reflexes of the right leg. Cerebellar coordination tests can not be elicited in the left leg because of weakness; the right side had accepted cerebellar coordination with no intension tremor, no overshooting, and no dyssynergia.

What is the interpretation?
The interpretation is LMN lesion of the left leg e.g. peripheral neuropathy.

Examination of the motor system of the lower limbs had shown that the patient was lying in dorsal decubitus posture with no wasting, weakness, or abnormal movements in both legs. The muscle tone of the left leg was slightly reduced (hypotonia) while the right leg had normal muscle tone with no hypertonia, hypotonia, or paratonia. The knee and ankle reflexes of both arms were normal with flexor planter response. Cerebellar coordination tests had shown the patient had intension tremor, overshooting, dyssynergia, and pendular jerk of the left side; the right side had shown normal cerebellar function.

What is the interpretation?
The interpretation is left cerebellar hemisphere lesion.

Examination of the motor system of the lower limbs had shown that the patient was lying in dorsal decubitus posture with no wasting or weakness. The muscle tone of both legs was increased (hypertonia) in the form of lead pipe rigidity. The knee and ankle reflexes of both legs were normal (grade 2); flexor planter response. The patient had slowed coordination in both legs.

What is the interpretation?
The interpretation is extrapyramidal lesion e.g. Parkinson's disease.

Examination of the motor system of the lower limbs had shown that the patient was lying in dorsal decubitus posture; both legs were extended and internally rotated. No involuntary movements can be noticed or elicited by flicking. There was normal muscle bulk of both legs. Increased muscles tones of both legs (hypertonia) in the form of spasticity; muscle power of both legs had shown that the flexor muscles were weak as compared with the extensor group (grade 0 vs grade 2). The knee and the ankle reflexes of both legs were three pluses (hyperreflexia) with sustained ankle clonus; extensor planter response of both legs. Cerebellar coordination tests can not be elicited in both legs because of weakness.

What is the interpretation?
The interpretation is UMN lesion affecting both legs (spastic paraperesis; see box 7 – 19).

CLINICAL CLUE

FOR UNDERGRADUATE AND POSTGRADUATE CANDIDATES

Trömner's sign is flexion of the thumb and index finger in response to tapping or flicking the volar surface of the distal phalanx of the middle finger, held partially flexed between the examiner's finger and thumb. This is an alternative method to Hoffmann's sign ('snapping' the distal phalanx) to elicit the finger flexor response. As in the latter, it is suggestive of a corticospinal tract (upper motor neurone) lesion above C5 or C6, especially if unilateral, although it may be observed in some normal individuals.

CLINICAL LOCALIZATION OF STROKE

Stroke is a syndrome characterized by the acute onset of a neurologic deficit that persists for at least 24 hours, reflects focal involvement of the central nervous system, and is the result of a disturbance of the cerebral circulation. Completed stroke describes a stroke in which the focal deficit persists and is not progressing. A stroke that is actively progressing as a direct consequence of the underlying vascular disorder (but not because of associated cerebral edema) or has done so in recent minutes is termed stroke in evolution or progressing stroke. Focal cerebral deficits that develop slowly (over weeks to months) are unlikely to be due to stroke and are more suggestive of tumor or inflammatory or degenerative disease. When symptoms and signs resolve completely after briefer periods (usually within 30 minutes), the term transient ischemic attack (TIA) is used. Recurrent transient ischemic attacks with identical clinical features are usually caused by thrombosis or embolism arising within the cerebral circulation while transient ischemic attacks that differ in character from event to event suggest recurrent emboli from a cardiac source. In some cases, deficits last for longer than 24 hours but resolve completely or almost completely within a few days; the term reversible ischemic neurological deficit (RIND) or minor stroke is sometimes used to describe these events. As their names imply, transient ischemic attacks (TIAs) and reversible ischemic neurologic deficits (RINDs) are uniquely associated with cerebral ischemia, as opposed to hemorrhage.

Features that distinguish anterior from posterior circulation (FIGURE 7 – 62) are:

Symptom or Sign	Anterior	Posterior
Aphasia	Yes	No
Diplopia	No	Yes
Vertigo	No	Yes
Drop attacks (sudden collapse without loss of consciousness)	No	Yes

Congenital cardiac anomalies associated with a pathologic communication between the right and left sides of the heart, such as atrial septal defect or patent foramen ovale, permit the passage of embolic material from the systemic venous circulation to the brain. Under these circumstances, venous thrombi can give rise to embolic stroke termed paradoxical embolization.

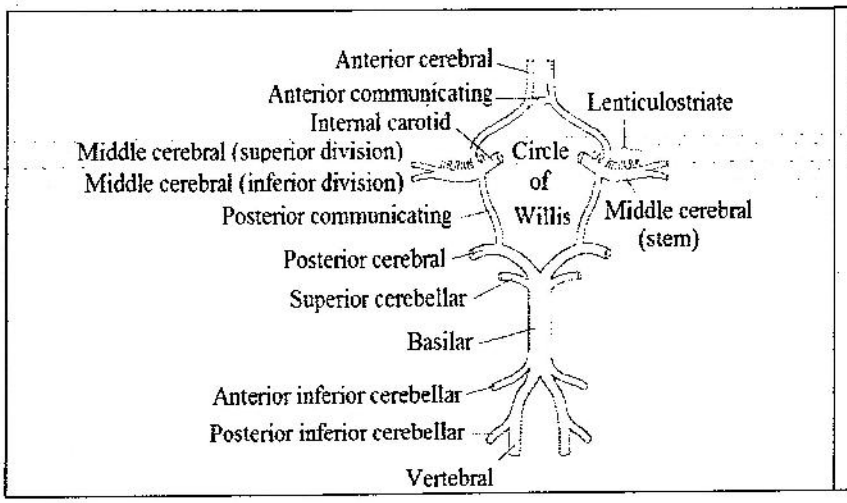


FIGURE 7 – 62. Arteries of the anterior (white) and posterior (blue) cerebral circulation in relation to the circle of Willis.

CLINICAL SYNDROME OF ANTERIOR CEREBRAL ARTERY OCCLUSION

Anterior cerebral artery strokes are uncommon, perhaps because emboli from the extracranial vessels or the heart are more apt to enter the larger-caliber middle cerebral artery, which receives the bulk of cerebral blood flow. There is a contralateral paralysis and sensory loss affecting the leg more than the arm. Voluntary control of micturition may be impaired because of failure to inhibit reflex bladder contractions, resulting in precipitate micturition.

CLINICAL SYNDROME OF MIDDLE CEREBRAL ARTERY OCCLUSION

The middle cerebral artery is the vessel most commonly involved in ischemic stroke. Depending on the site of involvement, several clinical syndromes can occur:

A. Superior division stroke results in contralateral hemiparesis that affects the face, hand, and arm but spares the leg; contralateral hemisensory deficit in the same distribution; but no homonymous hemianopia. If the dominant hemisphere is involved, these features are combined with Broca's (expressive) aphasia.

B. Inferior division stroke is less common in isolation and results in contralateral homonymous hemianopia that may be denser inferiorly; marked impairment of cortical sensory functions, such as graphesthesia and stereognosis on the contralateral side of the body; and disorders of spatial thought, including a lack of awareness that a deficit exists

(anosognosia), neglect of and failure to recognize the contralateral limbs, dressing and constructional apraxia. If the dominant hemisphere is involved, Wernicke's (receptive) aphasia occurs. With involvement of the nondominant hemisphere, an acute confusional state may occur.

C. Occlusion at the bifurcation or trifurcation of the middle cerebral artery involves a lesion situated at the point where the artery splits into two (superior and inferior) or three (superior, middle, and inferior) major divisions. This severe stroke syndrome combines the features of superior and inferior division stroke. Its clinical features include contralateral hemiparesis and hemisensory deficit involving the face and arm far more than the leg; homonymous hemianopia; and, if the dominant hemisphere is affected, global (combined expressive and receptive) aphasia.

D. Occlusion of the stem of the middle cerebral artery occurs proximal to the origin of the lenticulostriate branches. Because the entire territory of the artery is affected, this is the most devastating of middle cerebral artery strokes. The resulting clinical syndrome is similar to that seen following occlusion at the trifurcation except that, in addition, infarction of motor fibers in the internal capsule causes paralysis of the contralateral leg. The result is a contralateral hemiplegia and sensory loss affecting the face, hand, arm, and leg.

CLINICAL SYNDROME OF THE INTERNAL CAROTID ARTERY OCCLUSION

In about 15% of cases, progressive atherosclerotic occlusion of the internal carotid artery is preceded by premonitory TIAs or by transient monocular blindness caused by ipsilateral retinal artery ischemia. Carotid artery occlusion may be asymptomatic. Symptomatic occlusion results in a syndrome similar to that of middle cerebral artery stroke.

CLINICAL SYNDROME OF POSTERIOR CEREBRAL ARTERY OCCLUSION

Occlusion of a posterior cerebral artery produces homonymous hemianopia affecting the contralateral visual field with macular sparing because of the dual (middle and posterior cerebral artery) blood supply to the portion of the visual cortex representing the macula. In contrast to visual field defects from infarction in the middle cerebral artery territory, those caused by posterior cerebral artery occlusion may be denser superiorly.

CLINICAL SYNDROME OF BASILAR ARTERY OCCLUSION

Thrombotic occlusion of the basilar artery, a serious event that is often incompatible with survival, produces bilateral neurologic signs referable to involvement of multiple branch arteries. Occlusion of both vertebral arteries or of a lone unpaired vertebral artery produces a similar syndrome. Temporary occlusion of one or both vertebral arteries can also occur in relation to rotation of the head in patients with cervical spondylosis, leading to transient symptoms and signs of brainstem dysfunction. Emboli small enough to pass through the vertebral arteries into the larger basilar artery are usually arrested at the top of the basilar artery, where it bifurcates into the posterior cerebral arteries. The resulting reduction in blood flow to the ascending reticular formation of the midbrain and thalamus produces immediate loss or impairment of consciousness with unilateral or bilateral oculomotor (III) nerve palsies, and hemiplegia or quadriplegia with decerebrate or decorticate posturing.

CLINICAL SYNDROMES CAUSED BY LACUNAR INFARCTIONS

Small penetrating arteries located deep in the brain may become occluded as a result of changes in the vessel wall induced by chronic hypertension. The resulting lacunar infarcts are most common in deep nuclei of the brain (putamen, thalamus, caudate nucleus), the pons, and the posterior limb of the internal capsule. They occur in lesser numbers in the deep cerebral white matter, the anterior limb of the internal capsule, and the cerebellum. Because of their small size and their frequent location in relatively silent areas of the brain, many lacunar infarctions are not recognized clinically. Patients are usually presented with one of the following four syndromes:

I Pure motor hemiparesis

This consists of hemiparesis affecting the face, arm, and leg to a roughly equal extent, without an associated disturbance of sensation, vision, or language

II Pure sensory stroke

This is characterized by hemisensory loss

III Ataxic hemiparesis

In this syndrome, sometimes called ipsilateral ataxia and crural (leg) paresis, pure motor hemiparesis is combined with ataxia of the hemiparetic side and usually predominantly affects the leg

IV Dysarthria-clumsy hand syndrome

This consists of dysarthria, facial weakness, dysphagia, and mild weakness and clumsiness of the hand on the side of facial involvement

SENSORY SYSTEM EXAMINATION

Evaluating sensation is usually the most unreliable part of the examination, because it is subjective and is difficult to quantify. In the compliant patient, the sensory examination can be extremely helpful for the precise localization of a lesion. Try to fit the distribution of any sensory loss into a dermatome (due to a spinal cord or nerve root lesion), a sensory level (transverse spinal cord lesion) a single peripheral nerve territory, a peripheral neuropathy pattern (glove and stocks distribution), or a hemisensory loss (due to spinal cord or upper brainstem or thalamic lesion).

GENERAL RULE: THE EAXAMINATION DEPENDS ON THE ORDER

I. Test sensation according to the dermatomes

Test each dermatome as shown in FIGURE 7 - 63

II. Test for sensory level

A sensory level is a spinal cord level below which there is a marked decrease in sensation. Start distally and continue proximally if abnormal

III. Test for gloves and stocks distribution

Check sensation distally and ascend if abnormal

IV. Examine sensation (without specification)

Check sensation distally and ascend if abnormal

V. Check sensation of C6 and C7

Test these dermatomes as shown in FIGURE 7 - 63

VI. Examine the spinothalamic tract functions

Check light touch, pain, and temperature (as shown below)

VII. Examine the dorsal column system function

Check position sense and vibration (as shown below)

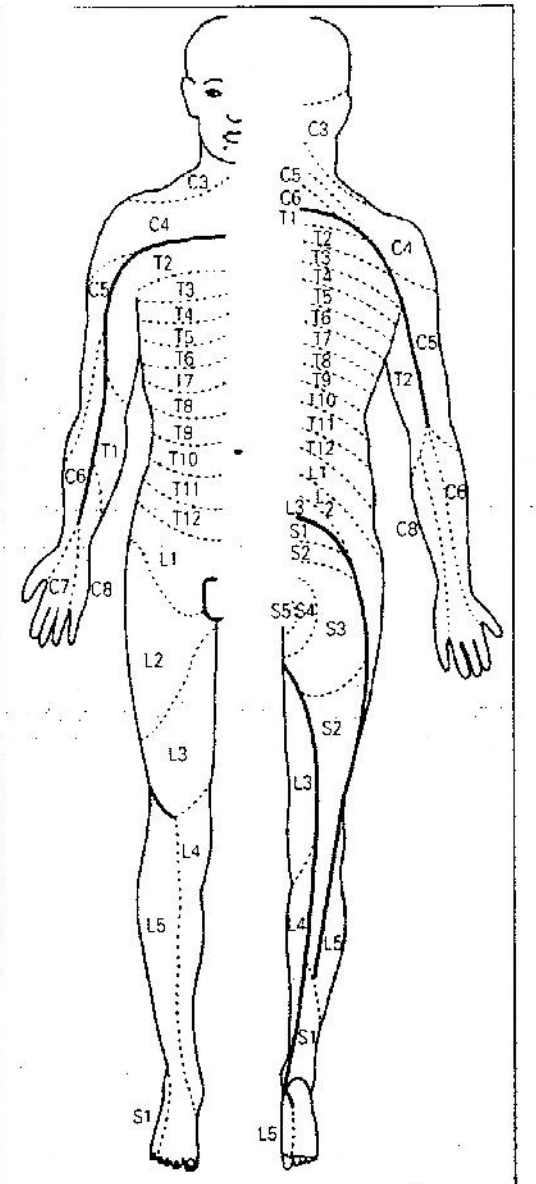


FIGURE 7 – 63. Distribution of the sensory spinal roots on the surface of the body (dermatomes).

Damage to the afferent nervous pathways conveying sensations of touch and pain produces either the negative sensation of numbness or positive symptoms, such as paresthesia and pain. The term paresthesias typically refers to tingling or pins-and-needles sensations but may include a wide variety of other abnormal sensations, except pain; it sometimes implies that the abnormal sensations are perceived spontaneously. The more general term dysesthesias denotes all types of abnormal sensations, including painful ones, regardless of whether a stimulus is evident. Another set of terms refers to sensory abnormalities found on examination. Anesthesia refers to the absence of touch sensation; hypesthesia to decreased sensitivity; and hyperesthesia to increased sensitivity. Similarly, analgesia refers to absence of pain sensation, hypalgesia to decreased sensitivity to pain, and hyperalgesia to increased sensitivity. Allodynia describes the situation in which a nonpainful stimulus, once perceived, is experienced as painful, even excruciating. An example is elicitation of a painful sensation by application of a vibrating tuning fork. Hyperpathia, a broad term, encompasses all the phenomena described by hyperesthesia, allodynia, and hyperalgesia.

The sensory examination consists of testing for the following:

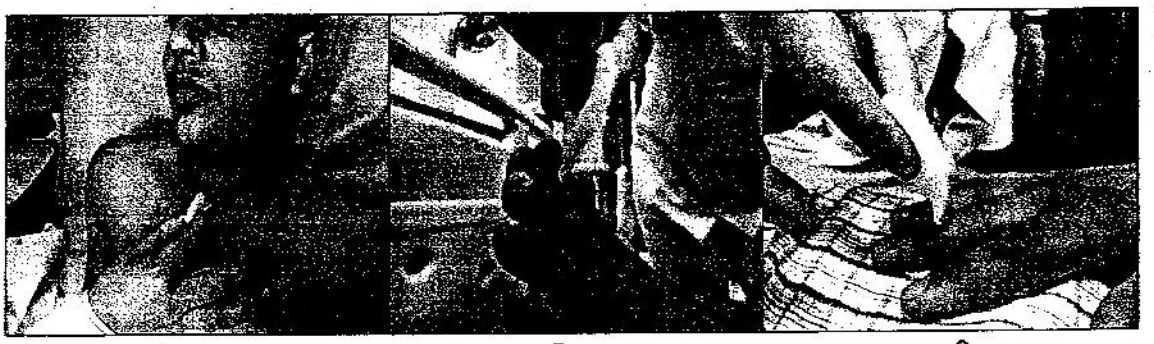
Small fiber (spinothalamic) function
- Light touch sensation
- Superficial pain sensation
- Temperature sensation

Large fiber (dorsal column) function
- Proprioception (joint position)
- Vibration sense

Cortical sensory function
- Stereognosis
- Graphesthesia
- Two-point discrimination
- Tactile localization
- Extinction

TEST LIGHT TOUCH (FIGURE 7 – 64)

Light touch is evaluated by lightly touching the patient with a wisp of cotton wool. Start testing pain sensation on the sternum as a base line. Ask the patient then to close his/her eyes and to tell you when the touch is felt. Try touching the patient on the toes/fingers. Make a dabbing stimulus and do not stroke the skin because this moves hair fibres. If sensation is normal, continue with the next test. If sensation is abnormal, work proximally until a sensory level can be determined. Test each dermatome comparing left and right sides. Anesthesia is absence of touch sensation, hypesthesia is a decreased sensitivity, and hyperesthesia is an increased sensitivity.

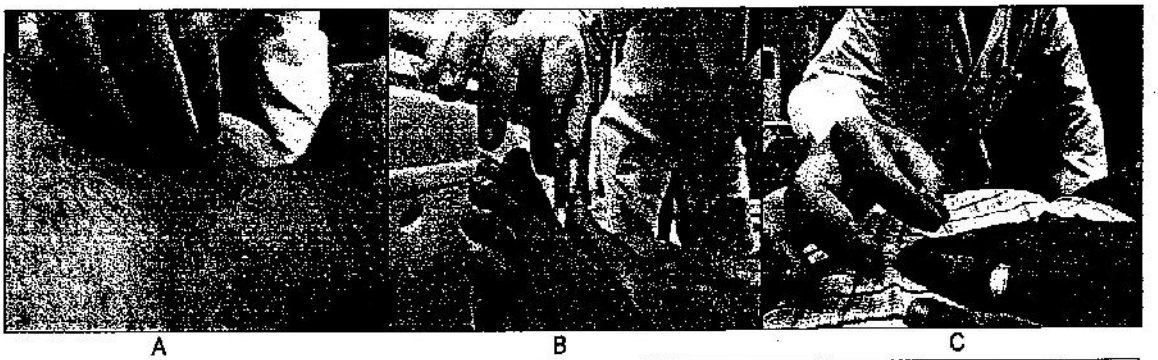


A B C

FIGURE 7 – 64. Test light touch over the sternum as a base line (A), then over the great toe (B) in the lower limbs, and over the fingers (C) in the upper limbs.

TEST SUPERFICIAL PAIN SENSATION (FIGURE 7 – 65)

Pain sensation is tested by using a new safety pin and asking the patient if it is felt. Start testing pain sensation on the sternum as a base line. Ask the patient to close his or her eyes and to tell you when the pain is felt. Try sensation on the toes/fingers. If the patient has no loss of sensation, proceed with the next examination. If there is a sensory loss to pain, continue proximally to determine the sensory level. Test each dermatome comparing left and right sides. Analgesia refers to absence of pain sensation, hypalgesia to decreased sensitivity to pain, and hyperalgesia to increase in sensitivity. To test whether or not the patient is malingerer, ask the patient to close his /her eyes, without pricking ask him/her about the pain of pricking.



A B C

FIGURE 7 – 65. Test light test over the sternum as a base line (A), then over the great toe (B) in the lower limbs, and over the fingers (C) in the upper limbs.

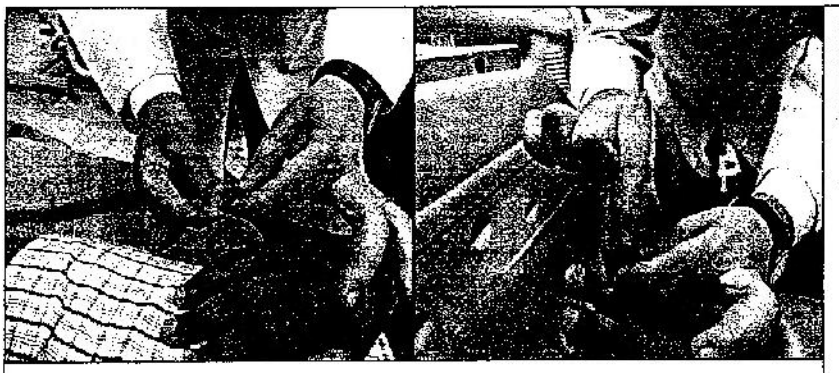
TEST TEMPERATURE SENSATION (NOT USUALLY PERFORMED)

This can be done in a similar fashion to the previous tests, using test tubes filled with hot (40-45 degrees) and cold (5-10 degrees) water.

TEST POSITION SENSE (FIGURE 7 – 66)

Position sense, or proprioception, is tested by moving the distal phalanx of little finger/big toe. Grasp the sides of the distal phalanx (so that the patient cannot be clued by the pressure exerted on the digit), and move the digit up while telling the patient (This is up). Move the distal phalanx then down and tell the patient (This is down). With the patient's eyes closed, move the distal phalanx up and down and finally stop and ask (What is this, up or down?). If no loss of position sense is detected, continue with the rest of the examination. If position sense is impaired, move proximally to test it at the ankle joint and if necessary, the knees and hips. In a similar fashion, test position in the little finger, moving proximally if indicated to the metacarpophalangeal joints, wrist, and elbow. As a rule, sense of position is lost before sense of movement, and the little finger is affected before the thumb.

Remember that three mistakes out of five trials indicate positional sense impairment.

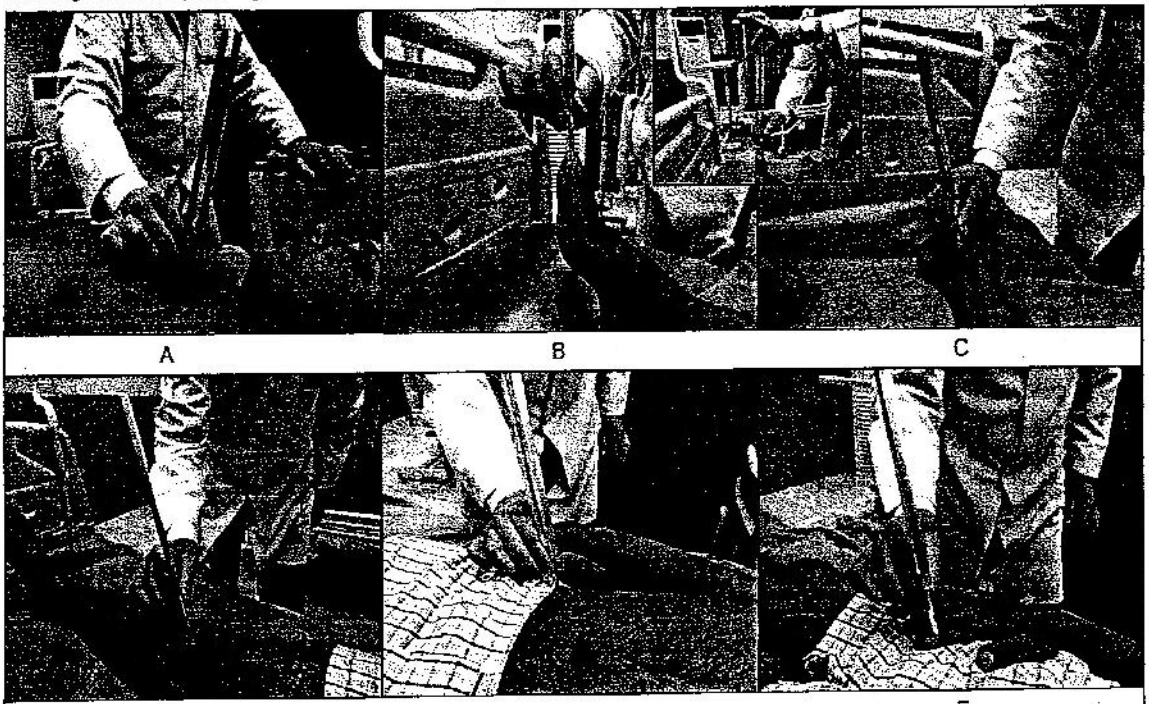


A B

FIGURE 7 – 66. Testing of the position sense in the little finger (A) and big toe (B).

TEST VIBRATION SENSE (MAINLY DESIGNED FOR POSTGRADUATE STUDENTS) (FIGURE 7 – 67)

Vibration sense is tested using a 128 Hz tuning fork. Strike a 128 Hz tuning fork confidently on your thenar eminence and demonstrate the vibration of the tuning fork on the patient's sternum as a base line and then place it on a bony prominence distally. Ask the patient to close the eyes, and then place the vibrating tuning fork on the distal interphalangeal joints of forefinger/on big toe. The patient should be able to describe a feeling of vibration. The candidate then deadens the tuning fork with the hand, and the patient should be able to say exactly when this occurs. Compare one side with the other. If the patient has no loss of sensation, proceed with the next examination. If vibration sense is reduced or absent, in the upper limbs test over the ulnar head at the wrist, then the elbows (over the olecranon) and then the shoulders to determine the level of abnormality. In the lower limbs, if vibration sense is absent on the great toe, try testing on the medial malleolus, on the patella, and then on the anterior superior iliac spine.



A B C

D E F

G H

FIGURE 7 – 67. Test vibration sensation over the sternum as a base line (A), over the great toe (B) and the way of deaden the fork. If abnormal, over the medial malleolus (C), over the patella (D), and over the ASIS (E) in the lower limbs; in the lower limbs, test over the fingers (F). If abnormal test over the ulna (G) and the olecranon, and over the shoulder (H).

INTERPRETATION OF THE SENSORY FINDINGS

The afferent sensory limb is important in the conscious appreciation of sensation. Nerve fibers carrying pain, light touch and temperature sensation enter the spinal cord and cross to the other side within one or two spinal segments. They ascend in the contralateral lateral spinothalamic tract, travel through the brain stem and the thalamus, and end in the postcentral gyrus of the parietal lobe, as illustrated in the FIGURE 7 – 68. Fibers carrying joint position and vibration sensation via medial lamniscal tract (dorsal column system) pass directly into the posterior columns and ascend in the fasciculi gracilis and cuneatus to their ipsilateral nuclei, cross in the medial lemniscus, synapse in the thalamus, and end in the postcentral gyrus of the parietal lobe.

A lesion on one side of the spinal cord that affects lateral spinothalamic tract results in elimination of pain and temperature sensation on the opposite side of spinal cord below the level of injury; a lesion of the dorsal column system in the spinal cord causes loss of position sense and vibration sensation on the same side of the body; a lesion above the level of the medulla oblongata causes loss of all sensory modalities on the opposite side of the body.

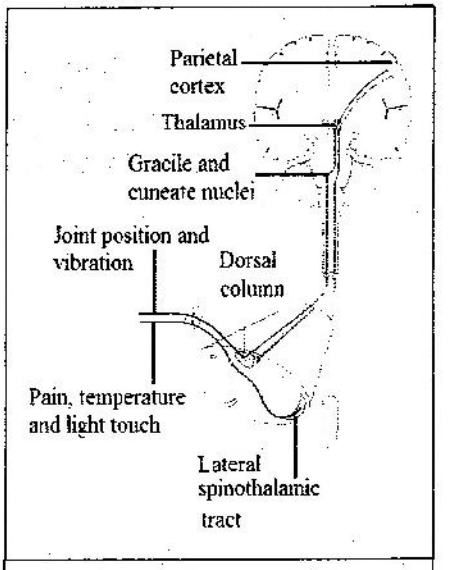


FIGURE 7 – 68. An illustration of the sensory system.

PATTERN OF SENSORY DISTURBANCE

The distribution of sensory loss (FIGURE 7 – 69) is often very useful in making a diagnosis when combined with the coexisting deficits of motor and/or cranial nerve function.

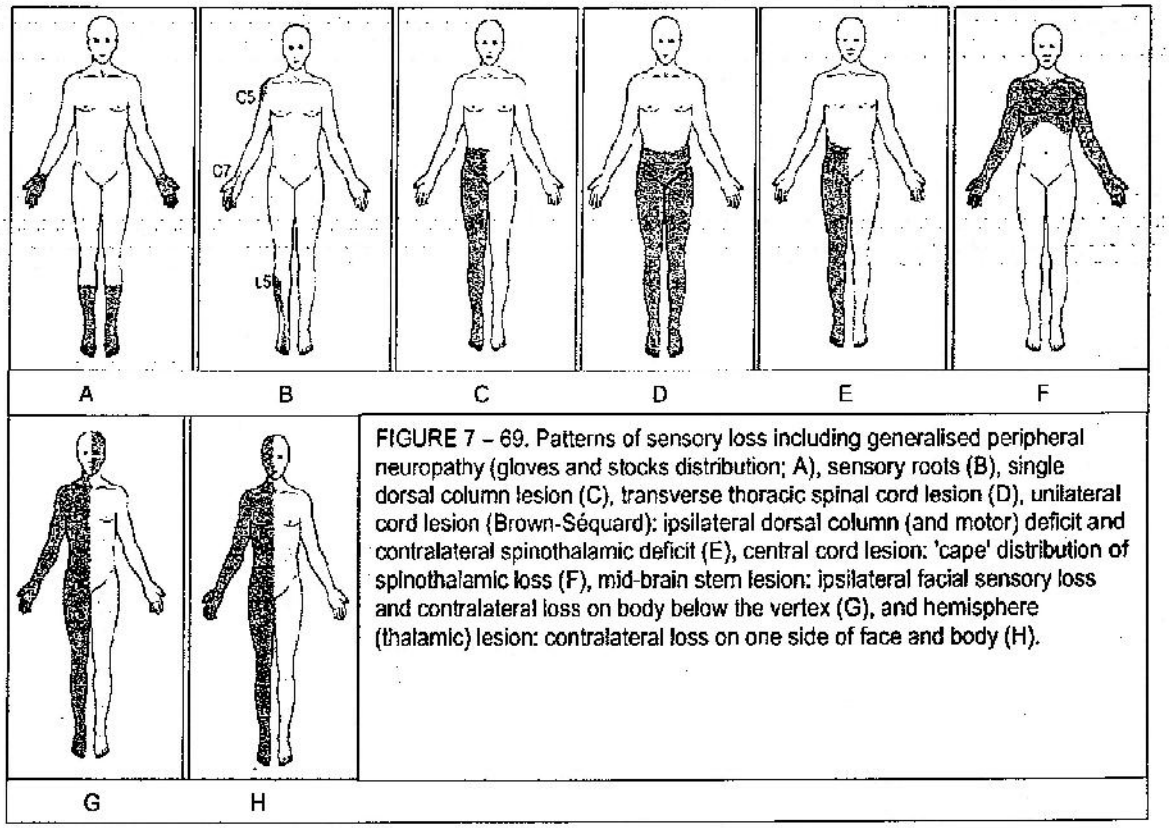


FIGURE 7 – 69. Patterns of sensory loss including generalised peripheral neuropathy (gloves and stocks distribution; A), sensory roots (B), single dorsal column lesion (C), transverse thoracic spinal cord lesion (D), unilateral cord lesion (Brown-Séquard): ipsilateral dorsal column (and motor) deficit and contralateral spinothalamic deficit (E), central cord lesion: 'cape' distribution of spinothalamic loss (F), mid-brain stem lesion: ipsilateral facial sensory loss and contralateral loss on body below the vertex (G), and hemisphere (thalamic) lesion: contralateral loss on one side of face and body (H).

t. Peripheral nerve lesions

In peripheral nerve lesions, the symptoms are usually of sensory loss and simple paresthesia (pins and needles). Single peripheral nerve lesions will, as expected, cause disturbance in the sensory distribution of that nerve. In patients with polyneuropathies, sensory loss is generally symmetric and is greater distally than proximally— as suggested by the term stocking-and-glove sensory loss (FIGURE 7 – 69 A) or by length-dependent neuropathy. As a general rule, the loss will have progressed almost to the knees before the hands are affected. Distal parts of the nerves are usually involved first because of their distance from the cell bodies. On examination there will be loss of all or selected (e.g. pain and touch) modalities.

II. Nerve root lesions (FIGURE 7 – 69 B)
Pain is more often a feature of lesions of nerve roots, within the spine or of the limb plexuses. It is often felt in the muscles innervated by a root, i.e. the myotome rather than the dermatome. The site of nerve root lesions may be deduced from the dermatomal pattern of sensory loss, although this is often smaller than would be expected because of the overlap of sensory territories.

III. Spinal cord lesions
Transverse lesions of the spinal cord produce loss of all modalities below that segmental level, although the level obtained clinically may vary by two or three segments (FIGURE 7 – 69 D). Very often, at the top of the area of sensory loss, there is a band of paresthesia or hyperesthesia. If the transverse lesion is vascular in origin (due to anterior spinal artery thrombosis), the posterior one-third of the spinal cord (and therefore the dorsal column modalities) may be spared. Lesions damaging one side of the spinal cord will produce sensory loss for spinothalamic modalities (pain and temperature) on the opposite side and for dorsal column modalities (joint position and vibration) on the same side as the lesion. This is the pattern seen in the Brown-Séquard syndrome (FIGURE 7 – 69 E). Lesions in the centre of the spinal cord (e.g. syringomyelia), spare the dorsal columns but affect the spinothalamic fibres crossing the cord from both sides over the length of the lesion (FIGURE 7 – 69 F; cape distribution). The sensory loss is therefore dissociated (in terms of the modalities affected) and suspended (in the sense that segments above and below the lesion are spared), often with reflex loss if afferent fibers of the reflex arc within the cord are affected.
There may be a lesion in the dorsal column alone (FIGURE 7 – 69 C), particularly in multiple sclerosis. This produces a characteristic unpleasant tight feeling over the limb involved and loss of proprioception that may severely affect the function of the limb without any loss of pin-prick or temperature sensation.

IV. Brain-stem lesions
The second-order neurons of the dorsal column sensory system cross the midline in the upper medulla to ascend through the brain stem. Here they lie just medial to the (already crossed) spinothalamic pathway. Brain-stem lesions can therefore cause sensory loss affecting all modalities of the contralateral side of the body (FIGURE 7 – 69 G; midbrain lesion). Sensory loss on the face due to brain-stem lesions is dependent upon the anatomy of the trigeminal fibres within the brain stem and may in a (balaclava helmet) distribution. Balaclava Helmet is a pattern of facial sensory loss resembling in distribution a balaclava helmet, involving the outer parts of the face but sparing the nose and mouth, may be seen with central brainstem lesions such as syringobulbia which progress upwards from the neck, such that the lowermost part of the spinal nucleus of the trigeminal nerve which serves the outer part of the face is involved whilst the upper part of the nucleus which serves the central part of the face is spared. This pattern of facial sensory impairment may also be known as onion peel or onion skin.

V. Thalamic and hemisphere lesions
Both the dorsal column and spinothalamic tracts end in the thalamus, relaying from there to the parietal cortex. In the thalamus, discrete lesions (as may occur in small lacunar strokes) can cause loss or impairment of all forms of sensation over the whole contralateral half of the body (FIGURE 7 – 69 H). Spontaneous pain, sometimes with a particularly unpleasant quality, may occur on the affected side. Patients may describe it as burning, tearing, knifelike, or stabbing, but often have difficulty characterizing it. Such a thalamic syndrome (Dejerine-Roussy syndrome) can also occasionally result from lesions of the white matter of the parietal lobe. Disease limited to the parietal sensory cortex impairs discriminative cortical sensory function on the whole opposite side of the body (FIGURE 7-69 H). Lesions in the sensory cortex have to be very small (and therefore affect only a restricted area of the body) to avoid affecting the motor tracts deeper in the hemispheres. With substantial lesions of the parietal cortex (as with large strokes) there is severe loss of proprioception and even conscious awareness of the existence of the affected limb (s). The resulting loss of function in the limb may be impossible to distinguish from paralysis.

Dissociated sensory loss refers to impairment of selected sensory modalities with preservation, or sparing, of others. It is usually an indication of an intramedullary spinal cord lesion. For example, focal central cord pathology such as syringomyelia will, in the early stages, selectively involve decussating fibres of the spinothalamic pathway within the ventral commissure, thus impairing pain and temperature sensation (often in a suspended, 'cape-like', 'bathing suit', 'vest-like', or cuirasse distribution), whilst the dorsal columns are spared, leaving proprioception intact. The anterior spinal artery syndrome also leaves the dorsal columns intact. Conversely, pathologies confined, largely or exclusively, to the dorsal columns (classically tabes dorsalis and subacute combined degeneration of the cord from vitamin B12 deficiency, but probably most commonly seen with compressive cervical myelopathy) impair proprioception, sometimes sufficient to produce sensory ataxia, whilst pain and temperature sensation is preserved. A double dissociation of sensory modalities on opposite sides of the trunk is seen in the Brown–Séquard syndrome.

DISEASES OF THE PERIPHERAL NERVOUS SYSTEM

Numerous inherited and acquired pathological processes may affect the nerve roots (radiculopathy), the nerve plexuses (plexopathy) and/or the individual nerves (neuropathy).

SPINAL ROOT LESIONS (RADICULOPATHY)

Spinal root lesion causes radiculopathy. A radiculopathy is a disorder of nerve roots, causing pain in a radicular distribution, paresthesia, sensory diminution or loss in the corresponding dermatome, and lower motor neurone type weakness with reflex diminution or loss in the corresponding myotome. Radiculopathies may be single or multiple (polyradiculopathy, e.g. cauda equina syndrome). Most radiculopathies are in the lumbosacral region (60–90%), followed by the cervical region (5–30%). Lesions of the spinal roots are most commonly caused by compression at or near their spinal exit foramina by prolapsed intervertebral discs or degenerative spinal disease. Spinal roots may also be infiltrated by spinal and paraspinal tumour masses and inflammatory or infective processes.

PLEXUS LESIONS (PLEXOPATHY)

Lesions confined to the brachial, lumbar, or sacral plexi may produce a constellation of motor and sensory signs (weakness, reflex diminution or loss, sensory loss) which cannot be ascribed to single or multiple roots (radiculopathy) or peripheral nerves (neuropathy). Lesions may involve the whole plexus (panplexopathy) or be partial, e.g. upper trunk of brachial plexus (C5–C6), producing 'waiter's tip' posture (as for C5/C6 root avulsion). Neurophysiological studies may be helpful in distinguishing plexopathy from radiculopathy: sensory nerve action potentials are reduced or absent in plexopathies because the lesion is located distal to the dorsal root ganglion, whereas sensory nerve action potentials are normal in radiculopathies because the lesion is proximal to the dorsal root ganglion. EMG shows sparing of paraspinal muscles in a plexopathy because the lesion is, by definition, distal to the origin of the dorsal primary rami. Coexistence of radiculopathy and plexopathy may invalidate these simple rules.

I. Brachial plexopathy

Trauma usually damages either the upper or the lower parts of the brachial plexus, according to the mechanics of the injury. The clinical features depend upon the anatomical site of the damage (BOX 7 – 20)

BOX 7 – 20. Clinical features of plexopathies

Site	Root	Affected muscles	Sensory loss
Upper plexus (Erb-Duchenne)	C5/6	Biceps, deltoid, spinati, rhomboids, brachioradialis (triceps, serratus anterior)	Patch over deltoid
Lower plexus(Déjerine-Klumpke)	C8/T1	All small hand muscles, claw hand (ulnar wrist flexors)	Ulnar border hand/forearm
Thoracic outlet syndrome	C8/T1	Small hand muscles, ulnar forearm	Ulnar border hand/forearm/upper arm

Lower parts of the brachial plexus are vulnerable to infiltration from breast or apical lung tumours (Pancoast tumour) and may be damaged by therapeutic irradiation. The lower parts of the plexus may also be compressed by anatomical anomalies at the thoracic outlet. An acute brachial plexopathy of probable inflammatory origin may present with 'neuralgic amyotrophy'. In this syndrome, a period of very severe shoulder pain precedes the appearance of a patchy upper brachial plexus lesion, often affecting the long thoracic nerve which produces winging of the scapula. Recovery occurs over months and is usually complete.

II. Lumbosacral plexopathy

Lumbosacral plexus lesions may be caused by neoplastic infiltration or compression by retroperitoneal hematoma in patients with a coagulopathy. A small vessel vasculopathy can produce a lumbar plexopathy, especially in elderly patients when it may be the presenting feature of type 2 diabetes mellitus ('diabetic amyotrophy'). This presents with painful wasting of the quadriceps with weakness of knee extension and adduction, and an absent knee reflex.

PERIPHERAL NERVE LESIONS (NEUROPATHY)

Cranial nerves 3-12 share the same tissue characteristics as peripheral nerves elsewhere and are subject to the same range of diseases (BOX 7 – 21). Nerve fibres of different types (motor, sensory or autonomic) may be variably involved. Disorders may be primarily directed at the axon, the myelin sheath (Schwann cells) or both. An acute or chronic peripheral nerve disorder may be focal (affecting a single nerve: mononeuropathy), multifocal (several nerves: mononeuropathy multiplex) or generalised (polyneuropathy). Motor nerve involvement produces features of a lower motor neuron lesion. Symptoms and signs of sensory nerve involvement depend on the type of sensory nerve involved. Autonomic fiber involvement may cause postural hypotension due to disruption of vasomotor control, sweating, cardiac rhythm, and gastrointestinal, bladder and sexual functions.

BOX 7 – 21. Causes of peripheral neuropathies	
Small-fiber sensory (painful with dissociated sensory loss)	
Hereditary sensory neuropathies (early) Diabetic small-fiber neuropathy Lepromatous leprosy	Amyloidosis HIV/ antiretroviral therapy neuropathy
Large-fiber sensory (ataxic-neuropathies)	
Sjögren's syndrome Vitamin B_{12} neuropathy Cisplatin neuropathy	Pyridoxine toxicity Friedreich's ataxia
Small- and large-fiber (global sensory loss)	
Diabetic sensory neuropathy Carcinomatous sensory neuropathy	Hereditary sensory neuropathies
Motor-predominant neuropathies	
Guillain – Barré syndrome Heritable motor-sensory neuropathies Acute intermittent porphyria	Diphtheritic neuropathy Lead neuropathy
Autonomic neuropathies	
Acute:	Chronic:
Botulism, porphyria, GBS, amiodarone, vincristine	Amyloid, diabetes, Sjögren's, Chagas, paraneoplastic

NERVE THICKENING

If there is evidence of a peripheral nerve lesion, peripheral neuropathy or a mononeuritis multiplex, palpate for thickened nerves e.g. median nerve at wrist or ulnar nerve at elbow that are most easily accessible. If nerves are thickened consider acromegaly, amyloidosis, chronic inflammatory demyelinating neuropathy, leprosy, hereditary motor and sensory neuropathy, diabetes mellitus, or neurofibromatosis.

CORTICAL SENSORY FUNCTION (NONROUTINE TESTS) (FIGURE 7 – 70)

Abnormalities of these sensory tests, in the presence of normal primary sensation (e.g. touch, position sense, etc) in an alert cooperative patient, signify a lesion of the parietal cortex or thalamocortical projections to the parietal lobe. If primary sensation is altered, these cortical discriminative functions usually will be abnormal also. Comparisons should always be made between analogous sites on the two sides of the body because the deficit with a specific parietal lesion is likely to be unilateral. The following tests are used to test cortical sensations

I. STEREOGNOSIS

Stereognosis refers to the ability to identify an object by feeling it. Ask the patient to close his/her eyes and place in the patient's hand a familiar object such as a coin, paper clip, key, pencil, or cotton ball, and ask the patient to tell you what it is. Normally a patient will manipulate it skillfully and identify it correctly. Test the other hand and compare the findings. Astereognosis refers to the inability to recognize objects placed in the hand.

II. NUMBER IDENTIFICATION (GRAPHTHESIA)

Ask the patient to close his/her eyes and with the blunt end of a pen or pencil, draw a large number in the patient's palm. A normal person can identify most such numbers. Inability to recognize numbers, termed agraphesthesia, suggests a lesion in the sensory cortex.

III. TWO POINTS DISCRIMINATION

Ask the patient to close his/her eyes. Using the two ends of an opened paper clip, or the sides of two pins, touch a finger pad in two places simultaneously. Be careful not to cause pain. Find the minimal distance at which the patient can discriminate one from two points (normally less than 3 mm on the finger pads). This test may be used on other parts of the body, but normal distances vary widely from one body region to another. In the tongue, it is 2 mm (the most sensitive area of the body), 11 mm at the back of hand, and 64 mm at the back. Lesions of the sensory cortex increase the distance between two recognizable points.

IV. POINT LOCALIZATION

Ask the patient to close his/her eyes and briefly touch a point on the patient's skin. Then ask the patient to open both eyes and point to the place touched. Normal person can do so accurately. Lesions of the sensory cortex impair the ability to localize points accurately.

V. EXTINCTION

Have the patient close his or her eyes and identify where your touch was felt. Touch the patient's dorsum of both hands. The patient is then asked, (Where did I touch you?). Normally, patients have no problem identifying both areas. A patient with a lesion in the parietal lobe may feel the individual touches but may (extinguish) the sensation on the side contralateral to the side of the lesion. This is the phenomenon termed extinction.

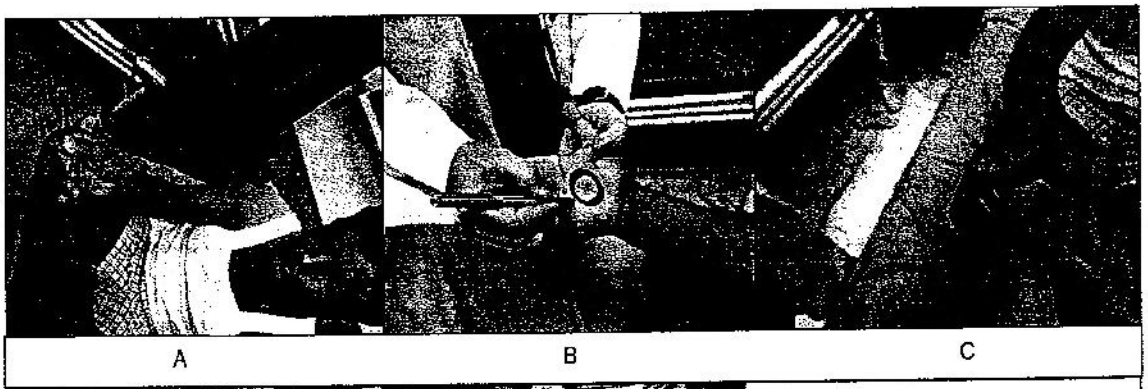


FIGURE 7 – 70. Cortical sensory function including stereognosis (A), graphesthesia (B), two points discrimination (C), point localization (D), and extinction (E).

EXAMPLES

Examine the sensory system of this patient
Examination of the sensory system had shown that the patient had intact light touch sensation, superficial pain sensation, joint position sense, and vibration sensation.

What is the interpretation?
The interpretation is normal sensory system examination.

Examination of the sensory system had shown that the patient had lost light touch sensation and superficial pain sensation in the distal parts of both arms and both legs involving the whole hands and the whole feet up to the mid point of the shins while joint position sense and vibration sensation were intact.

What is the interpretation?
The interpretation is peripheral neuropathy in the form of gloves and stocking distribution e.g. diabetes mellitus.

Examination of the sensory system had shown that the patient had lost position sense in the distal interphalangeal joints, the metacarpophalangeal joints, and the wrist joints of both arms as well as in the great toes and the ankle joints of both legs; position sense of both knees and both elbows were intact. Examination had also shown loss of vibration sensation over the distal interphalangeal joints and over the ulnar process in the upper limbs as well as over the great toes and medial malleoli in the lower limbs; vibration sensation was intact at higher levels. The light touch sensation and superficial pain sensation were intact.

What is the interpretation?
The interpretation is peripheral neuropathy in the form of gloves and stocking distribution e.g. Sjögren's syndrome.

Examination of the sensory system had shown that the patient had lost all the sensory modalities in the distal parts of both arms and legs.

EXAMPLES

What is the interpretation?
The interpretation is peripheral neuropathy in the form of gloves and stocking distribution e.g. diabetes mellitus.

Examination of the sensory system had shown that the patient had lost position sense in the great toe, ankle joint, knee joint, and hip joint of the right leg as well as loss of vibration sensation over the great toe, medial malleolus, patella, and anterior superior iliac spine of the right leg also; position sense and vibration sensation were intact in the left leg. The light touch sensation and superficial pain sensation were intact.

What is the interpretation?
The interpretation is right dorsal column lesion e.g. multiple sclerosis.

Examination of the sensory system had shown that the patient had lost all the sensory modalities in the legs up to T10; T11 and T12 had an increased light touch sensation. The arms had intact sensory function.

What is the interpretation?
The interpretation is transverse spinal cord lesion e.g. trauma.

Examination of the sensory system had shown that the patient had lost light touch sensation and pain sensation in the legs up to T10; T11 and T12 had an increased light touch sensation. The patient had normal vibration sensation and position sense. The arms had intact sensory function.

What is the interpretation?
The interpretation is anterior transverse spinal cord lesion e.g. anterior spinal artery thrombosis

Examination of the sensory system had shown that the patient had lost light touch sensation and pain sensation in the right leg up to T10 with loss of position sense and vibration sensation over the left leg. The arms had intact sensory function.

What is the interpretation?
The interpretation is left sided spinal cord lesion (Brown Séquard syndrome).

Examination of the sensory system had shown that the patient had lost all the sensory modalities over the right side of the body while the left side had intact sensory function.

What is the interpretation?
The interpretation is left thalamic lesion or brain stem lesion.
How can you further localize the lesion?
I can finely further localize the lesion if the trigeminal nerve sensation is examined.

Examination of the sensory system had shown that the patient had intact sensation over both sides of the body; the patient had lost the cortical sensory modalities over the right side of the body in the form of astereognosis, agraphthesia, inability to localize the point etc with intact cortical function in the left side.

What is the interpretation?
The interpretation is left parietal cortical lesion.

EXAMINATION OF THE PERIPHERAL NERVES (FOR POSTGRADUATE STUDENTS)

EXAMINATION OF THE PERIPHERAL NERVES OF THE UPPER LIMBS

I. MEDIAN NERVE (C6-T1)

The median nerve is formed in the axilla and then descends down the medial side of the arm in close association with the brachial artery to the cubital fossa. From there, the median nerve enters the forearm and gives off the anterior interosseous nerve. It then courses deep to the flexor retinaculum at the wrist (carpal tunnel) to reach the hand. This nerve contains the motor supply to all the muscles on the front of the forearm except the flexor carpi ulnaris. It also supplies the following short muscles of the hand (LOFA)-the lateral two lumbricals, opponens pollicis, in many people the flexor pollicis brevis, and abductor pollicis brevis. The examination sequence of the median nerve includes:

1 Weakness and wasting of the thenar eminence (implying wasting of the lateral two lumricals)

2 Lesion at the wrist (carpal tunnel)

Use the pen-touching test to assess for weakness of the abductor pollicis brevis (FIGURE 7 – 71 A). Ask the patient to lay the hand flat, palm upwards on the table, and attempt to abduct the thumb vertically to touch the examiner's pen held above it. This may be impossible if there is median nerve palsy at the wrist or above. The Opponens pollicis is tested (FIGURE 7 – 71 B) when the thumb is crossed over the palm to touch the little finger against resistance.

3 Lesion in the cubital fossa

Ochsner's clasping test is performed by asking the patient to clasp the hands firmly together (FIGURE 7 – 71 C), the index finger on the affected side fails to flex with a lesion in the cubital fossa or higher (FIGURE 7 – 71 D).

FIGURE 7 – 71. Testing the median nerve including the pen touching test (A), opponens pollicis test (B), and Ochsner's clasping test (C).

4 For the sensory component of the median nerve

The median nerve is the sensory supply to the thumb, anterior index and middle fingers as well as the radial side of the ring finger. Test pinprick sensation over the hand as shown in FIGURE 7 – 72. Remember that the palm is spared in median nerve lesions in the carpal tunnel while it will be involved in lesion above the wrist. The sensory loss is most severe at the tip of the index finger.

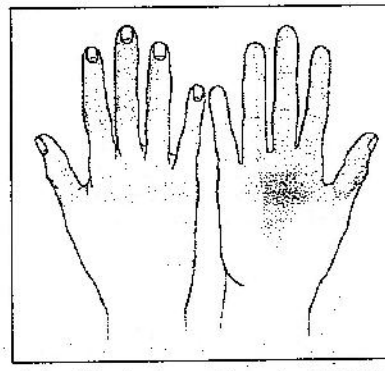


FIGURE 7 – 72. Sensory distribution of the median nerve.

5 Stretch tests for carpal tunnel syndrome

The median nerve may be compressed at the wrist within the carpal tunnel, resulting in the carpal tunnel syndrome (CTS). The principal symptom of CTS is a sensation of nocturnal paresthesias in the hand. The patient often awakens

and feels the need to shake out the hand. With increased severity, there may be intermittent pins and needles paresthesias in the hands during activities such as driving a car or even continuous numbness in several fingertips. When continuous sensory symptoms are present, the clinical finding of a sensory disturbance "splitting" the fourth digit is helpful in defining a median nerve lesion. Signs of mechanical irritability of a nerve, when elicited properly, are helpful. In particular, induction of tingling or brief electrical sensations in the tips of any of the second through fourth digits with tapping of the wrist over the median nerve (Tinel's sign) is highly suggestive of CTS. Phalen's sign (sensory symptoms within 1 minute of sustained wrist flexion) is most helpful when the symptoms include paresthesias in the sensory distribution of the median nerve as may produce hyperextension of the wrist (reversed Phalen's test). When these maneuvers produce wrist pain alone, or vague sensory symptoms, they should not be interpreted as independent evidence of median nerve compression. These stretch tests are shown in FIGURE 7 – 73.

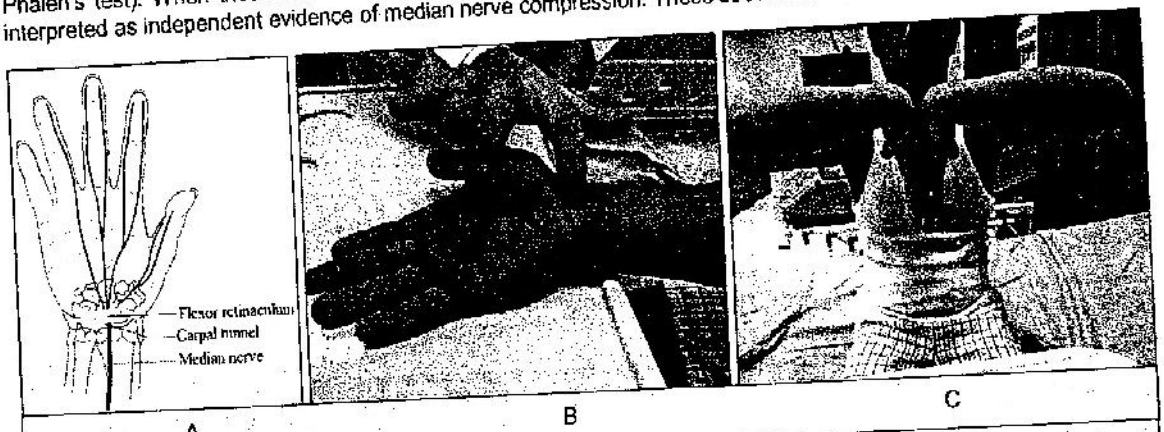


FIGURE 7 – 73. Anatomy of the carpal tunnel (A) and the stretch tests used to elicit tingling or electric sensation in the hands. These tests including Tinel's sign (B), Phalen's sign (C), and reversed Phalen's test (D).

II. RADIAL NERVE (C5-C8)

This is the motor nerve supplying the triceps and brachioradialis and the extensor muscles of the hand. The characteristic deformity that results from radial nerve injury is wrist drop. To demonstrate this, if it is not already obvious, get the patient to flex the elbow, pronate the forearm and extend the wrist and fingers. If a lesion occurs above the upper third of the upper arm the triceps muscle is also affected. Therefore, test elbow extension, which will be absent if the lesion, is high.

Test sensation using a pin over the area of the anatomical snuff box. Sensation here is lost with a radial nerve lesion before the bifurcation into posterior interosseous and superficial radial nerves at the elbow (FIGURE 7 - 74).

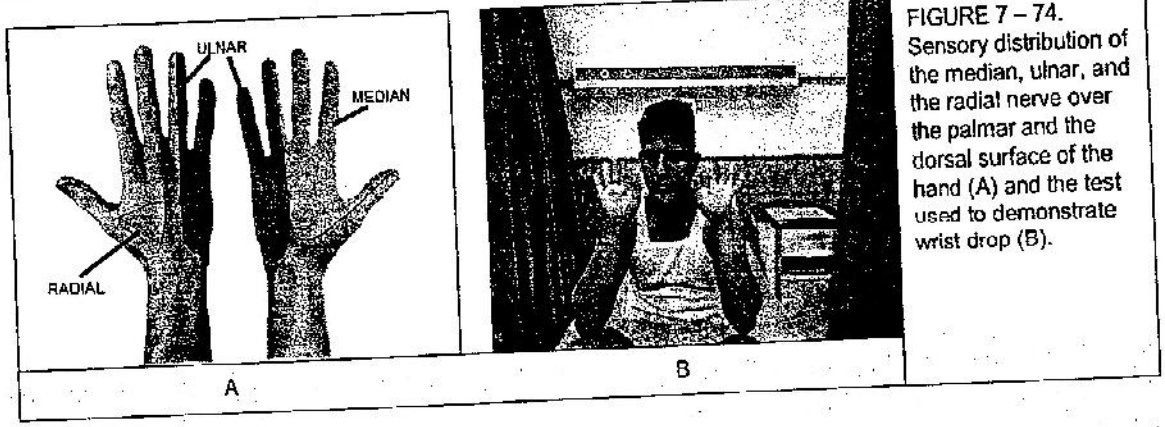


FIGURE 7 – 74. Sensory distribution of the median, ulnar, and the radial nerve over the palmar and the dorsal surface of the hand (A) and the test used to demonstrate wrist drop (B).

III. ULNAR NERVE
(C8-T1)
This nerve contains the motor supply to all the small muscles of the hand (except the LOFA muscles), flexor carpi ulnaris and the ulnar half of flexor digitorum profundus. The examination sequence includes:

1 Lesion at the wrist

Look for wasting and paralysis of the intrinsic hand muscles (except lateral two lumbricals) and of the hypothenar muscles. Look also for partial clawing of the little and ring fingers (a claw-like hand). Clawing (BOX 7 – 22) is hyperextension at the metacarpophalangeal joints and flexion of the interphalangeal joints. Note that clawing is more pronounced with an ulnar nerve lesion at the wrist, as a lesion at or above the elbow also causes loss of the flexor digitorum profundus, and therefore less flexion of the interphalangeal joints. This is the 'ulnar nerve paradox', in that a more distal lesion causes greater deformity

Froment's sign is performed by asking the patient to grasps a piece of paper between the thumb and lateral aspect of forefinger with each hand as shown in Figure 7 – 75 A. The affected thumb is unable to adduct so will flex instead as shown in Figure 7 – 75 B

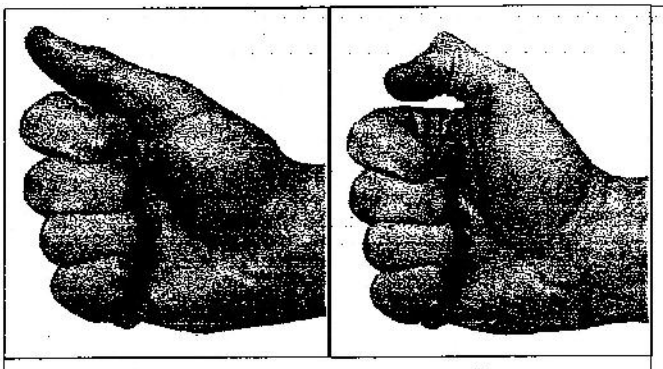
2 Lesion at the elbow

The manifestations will be as above but with less clawing. There will also be radial deviation of the wrist

3 The manifestations will be as above but with less clawing. There will also be radial deviation of the wrist

BOX 7 – 22. Causes of true claw hands

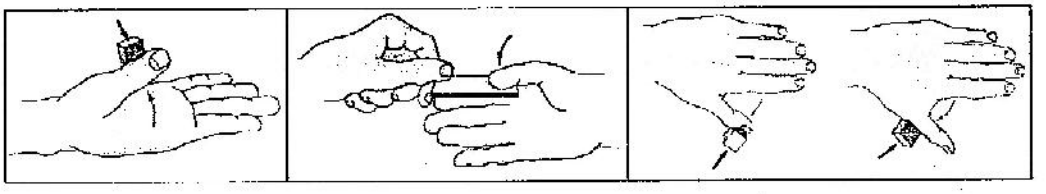
Ulnar and median nerve lesion	Other neurological disease e.g. syringomyelia, polio
Brachial plexus lesion (C8-T1)	Rheumatoid arthritis (advanced untreated disease)



A B

FIGURE 7 – 65. Froment's sign is performed by asking the patient to grasps a piece of paper between the thumb and lateral aspect of forefinger with each hand (A). The affected thumb is unable to adduct so will flex instead (B).

FOR POSTGRADUATE DOCTORS
EXAMINE THE MEDIAN, RADIAL, AND ULNAR NERVES IN THE THUMB ONLY
Test I: Use the pen-touching test to assess for weakness of the abductor pollicis brevis supplied by the median nerve (upper left view).
Test II: Use Froment's test to assess for weakness of adductor pollicis supplied by the ulnar nerve (upper right view).
Test III: The thumb is extended at the interphalangeal joint against resistance. This test is used to assess weakness in extensor pollicis longus supplied by the radial nerve (lower left view).



EXAMINATION OF THE PERIPHERAL NERVES OF THE LOWER LIMBS

I. LATERAL CUTANEOUS NERVE OF THE THIGH

Test for sensory loss as in Figure (FIGURE 7 – 76). A lesion of this nerve usually occurs because of entrapment between the inguinal ligament and the anterior superior iliac spine. It causes a sensory loss over the lateral aspect of the thigh with no motor loss detectable. If painful, it is called meralgia paraesthetica.

II. FEMORAL NERVE (L2, L3, L4)

This is the motor supply for the quadriceps muscles. Test for weakness of knee extension (quadriceps paralysis). Hip flexion weakness is only slight, and adductor strength is preserved. The knee jerk is absent. The sensory loss involves the inner aspect of the thigh and leg (FIGURE 7 – 76).

III. COMMON PERONEAL NERVE (L4, L5, S1)

This is a major terminal branch of the sciatic nerve. It supplies the anterior and lateral compartment muscles of the leg (FIGURE 7 – 76). On inspection one may notice a foot drop (BOX 7 – 23). Test for weakness of dorsiflexion and eversion. Test the reflexes which will all be intact. Test for sensory loss over the area in FIGURE 7 – 76. There is only minimal sensory loss over the lateral aspect of the dorsum of the foot. Note that these findings can be confused with an L5 root lesion, but the latter includes weakness of knee flexion and loss of foot inversion, as well as sensory loss in the L5 distribution.

FIGURE 7 – 76. Sensory distribution of the peripheral nerves in the lower limbs including lateral cutaneous nerve (A), femoral nerve (B), common peroneal nerve (C and D), and sciatic nerve (E and F).

BOX 7 – 23. Causes of foot drop

Common peroneal nerve or sciatic nerve palsy	Distal myopathy
Lumbosacral plexus lesion	Motor neurone disease
L4, L5 root lesion	Stroke-anterior cerebral artery or lacunar syndrome ('ataxic hemiparesis')
Peripheral motor neuropathy	

IV. SCIATIC NERVE (L4, L5, S1, S2, S3).

This nerve supplies all the muscles below the knee and the hamstrings (FIGURE 7 – 76). Test for loss of power below the knee resulting in a foot drop (plantar flexed foot) and for weakness of knee flexion. Test the reflexes: with a sciatic nerve lesion the knee jerk is intact but the ankle jerk and plantar response are absent. Test the sensation on the posterior thigh, lateral and posterior calf, and on the foot (lost with a proximal nerve lesion). Stretch tests for sciatica include:

1 The straight leg–raising (SLR)

With the patient supine, passive flexion of the extended leg at the hip stretches the L5 and S1 nerve roots and the sciatic nerve (straight leg–raising maneuver). Passive dorsiflexion of the foot during the maneuver adds to the stretch (Lasègue's test). While flexion to at least 80° is normally possible without causing pain, many patients normally report a tight, stretching sensation in the hamstring muscles unrelated to back pain. The straight leg–raising (SLR) test is positive if the maneuver reproduces the patient's usual back or limb pain (FIGURE 7 – 77 A).

2 Sitting knee extension test

Eliciting the SLR sign in the sitting position can help determine if the finding is reproducible. The patient may describe pain in the low back, buttocks, posterior thigh, or lower leg, but the key feature is reproduction of the patient's usual pain (FIGURE 7 – 77 B)

3 The crossed SLR sign

The crossed SLR sign is positive when flexion of one leg reproduces the usual pain in the opposite leg or buttocks. The crossed SLR sign is less sensitive but more specific for disk herniation than the SLR sign. The nerve or nerve root lesion is always on the side of the pain

4 The reverse SLR sign

The reverse SLR sign is elicited by standing the patient next to the examination table and passively extending each leg with the knee fully extended. This maneuver, which stretches the L2-L4 nerve roots, lumbosacral plexus, and femoral nerve, is considered positive if the patient's usual back or limb pain is reproduced

FIGURE 7 – 77. Stretch tests of the sciatic nerve including straight leg raising test (A), knee extension test (B), and crossed straight leg raising test (C).

EXAMINATION OF THE AUTONOMIC NERVOUS SYSTEM (MAINLY FOR POSTGRADUATE STUDENTS)

The preganglionic neurons of the parasympathetic nervous system leave the central nervous system in 3rd, 7th, 9th, and 10th cranial nerves as well as 2nd and 3rd sacral nerves, while the preganglionic neurons of the sympathetic nervous system exit the spinal cord between the first thoracic and the second lumbar segments. The postganglionic neurons, located in ganglia outside the CNS, give rise to the postganglionic autonomic nerves that innervate organs and tissues throughout the body. Acetylcholine (ACh) is the preganglionic neurotransmitter for both divisions of the ANS as well as the postganglionic neurotransmitter of the parasympathetic neurons. Norepinephrine (NE) is the neurotransmitter of the postganglionic sympathetic neurons, except for cholinergic neurons innervating the eccrine sweat glands. Disorders of the ANS may result from pathology of either the CNS or the PNS (BOX 7 – 24)

BOX 7 – 24. Causes of autonomic dysfunction

Diabetes mellitus	Craniopharyngioma
Guillain – Barré syndrome	Acute/subacute autonomic neuropathies (Botulism, porphyria, GBS, amiodarone, Vincristine)
Parkinson's disease, Multiple system atrophy (MSA), and Shy – Drager syndrome	Chronic peripheral autonomic neuropathies (Amyloid, diabetes, Sjögren's, Chagas, paraneoplastic)
Infection: HIV, Chagas disease, neurosyphilis	
Drugs: antihypertensive and diuretics	

AUTONOMIC TESTING

Autonomic function tests are helpful when the history and examination findings are inconclusive, to detect subclinical involvement, or to follow the course of an autonomic disorder. A combination of tests is usually necessary, because certain ones are particularly sensitive to abnormalities of sympathetic function and others to parasympathetic or baroreceptor afferent function. These tests include:

I. HEART RATE VARIATION WITH DEEP BREATHING

The normal heart rate varies with respiration (respiratory sinus arrhythmia). This is dependent on an intact parasympathetic system. The ECG is recorded while the patient breathes at a regular rate of 6 to 12 per minute. Normally, the heart rate varies by as many as 15 beats per minute or even more between expiration and inspiration; differences of less than 10 beats per minute may be abnormal. However, interpretation of results requires comparison of test data with results from normal individuals of the same age collected under the same test conditions. A more accurate test of vagal function is the measurement of the ratio of the longest R–R interval during forceful slow expiration to the shortest R–R interval during inspiration, which allows the derivation of an expiration-inspiration (E:I) ratio. Always, the results of these tests must be compared with those obtained in normal individuals of the same age.

II. VALSALVA RESPONSE

In the Valsalva maneuver, the supine subject exhales against a closed glottis for 10 to 15 s, creating a markedly positive intrathoracic pressure. The sharp reduction in venous return to the heart causes a drop in cardiac output and in blood pressure; the response on baroreceptors is to cause a reflex tachycardia and, to a lesser extent, peripheral vasoconstriction. With release of intrathoracic pressure, the venous return, stroke volume, and blood pressure rise to higher than normal levels; reflex parasympathetic influence then predominates and a bradycardia results. Failure of the heart rate to increase during the positive intrathoracic pressure phase of the Valsalva maneuver points to sympathetic dysfunction, and failure of the rate to slow during the period of blood pressure overshoot points to a parasympathetic disturbance.

III. RESPONSE OF BLOOD PRESSURE AND HEART RATE TO CHANGES IN POSTURE

It is stated that in changing from the recumbent to the standing position, a fall of more than 20 mmHg systolic and 15 mmHg diastolic is abnormal. It should be stressed that the arm on which the cuff is placed must be held horizontally when standing, so that the decline in arm pressure will not be obscured by the added hydrostatic pressure.

The main cause of an orthostatic drop in blood pressure is hypovolemia or autonomic dysfunction which can be discriminated via heart rate response. The failure of the heart rate to rise in response to the drop in blood pressure with standing is the simplest bedside indicator of vagal nerve dysfunction.

IV. PUPILLARY REACTION

The topical application of pharmacologic agents is particularly useful in evaluating pupillary denervation. Part of the rationale behind these special tests is "Cannon's law," or the phenomenon of denervation hypersensitivity, in which an effector organ, 2 to 3 weeks after denervation, becomes hypersensitive to its particular neurotransmitter substance and related drugs. The instillation of a 1:1000 solution of epinephrine into the conjunctival sac has no effect on the normal pupil but will cause the sympathetically denervated pupil to dilate (3 drops instilled three times at 1-min intervals). The pupillary size is checked after 15, 30, and 45 min. As a rule, hypersensitivity to epinephrine is greater with lesions of postganglionic fibers than of preganglionic fibers. If denervation is incomplete, the hypersensitivity phenomenon may not be demonstrable. Also, in lesions that involve central sympathetic pathways, the pupil rarely reacts to this test. More reliable as a test of sympathetic denervation is the topical application into the conjunctival sac of a 4 to 10% cocaine solution that potentiates the effects of NE by preventing its uptake. The test should be carried out as described above. A normal response to cocaine consists of pupillary dilatation. In sympathetic denervation caused by lesions of the post- or preganglionic fibers, no change in pupillary size occurs, since no transmitter

substance is available. The reason for lack of response in chronic preganglionic lesions is presumed to be a depletion of NE in the postganglionic fibers. In cases of central sympathetic lesions, slight myderiasis occurs.

V. TESTS OF BLADDER, GASTROINTESTINAL, AND PENILE ERECTILE FUNCTION

Bladder function is best assessed by the cystourethrogram. A simple way of determining bladder atony (prostatic obstruction and overdistention having been excluded) is to measure the residual urine (by catheterization of the bladder) immediately after voluntary voiding or to estimate its volume by ultrasound imaging.

Disorders of gastrointestinal motility are readily demonstrated radiologically. In dysautonomic states, a barium swallow may disclose a number of abnormalities, including atonic dilatation of the esophagus, gastric atony and distention, delayed gastric emptying time, and a characteristic small bowel pattern consisting of an increase in frequency and amplitude of peristaltic waves and rapid intestinal transit. A barium enema may demonstrate colonic distention and a decrease in propulsive activity. Sophisticated manometric techniques are now available for the measurement of gastrointestinal motility, especially in the esophagus.

Nocturnal penile tumescence is recorded in many sleep laboratories and may be used as an ancillary test of sacral autonomic (parasympathetic) innervation.

GAIT EXAMINATION

Seeing a patient walk can be very revealing for neurological diagnosis and is an important element of assessing disability. The examination is usually conducted through three steps. These are:

STEP I	TEST FOR PRONATOR DRIFT
STEP II	STANCE
STEP III	GAIT

TEST FOR PRONATOR DRIFT

Ask the patient to sit (or stand if he/she is able) over the edge of the bed if this is possible. Next ask the patient to hold out both hands, palms upward, with the arms extended and the eyes closed for 20 – 30 seconds (FIGURE 7 – 78). Watch the arms for evidence of drifting (movement of one or both arms from the initial neutral position). There are only three causes for drift of the arms:

A Upper motor neurone (pyramidal) weakness

The drift of the affected limb(s) here is due to muscle weakness and tends to be in a downward direction. The drifting typically starts distally with the fingers and spreads proximally. There may be slow pronation of the wrist and flexion of the fingers and elbow. This pronator drift is due to contralateral corticospinal tract lesion.

B Cerebellar disease

The drift here is usually upwards and is due to ipsilateral cerebellar hemispheric lesion. It also includes slow pronation of the wrist and elbow

C Loss of proprioception

The drift here (pseudoathetosis) is really a searching movement and usually affects only the fingers. It is due to loss of joint position sense and can be in any direction

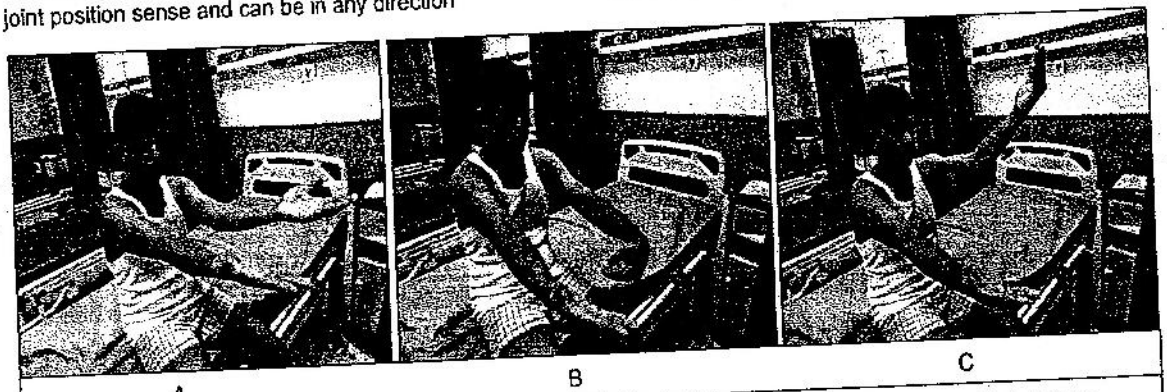


FIGURE 7 – 78. The technique of pronator drift (A), pyramidal drift (B), cerebellar drift (C), and drift due to loss of proprioception (D).

STANCE

Ask the patient to stand up straight, with the feet close together (preferably bare) and eyes open (FIGURE 7 – 79). Swaying or lurching with the eyes open suggests a cerebellar ataxia. If the patient had no cerebellar ataxia, ask him/her after that to close his eyes (Romberg's test). Repeatedly falling is a positive result and indicates sensory ataxia. Patients with sensory ataxia and some with vestibular ataxia are ultimately able to stand with the feet together, compensating for the loss of one source of sensory input (proprioceptive or labyrinthine) with another (visual). This

compensation is demonstrated when the patient closes the eyes, eliminating visual cues. With sensory or vestibular disorders, unsteadiness increases and results in falling (Romberg sign). With a vestibular lesion, the tendency is to fall toward the side of the lesion. Patients with cerebellar ataxia are unable to compensate for their deficit by using visual input and are unstable to stand straight on their feet whether the eyes are open or closed.

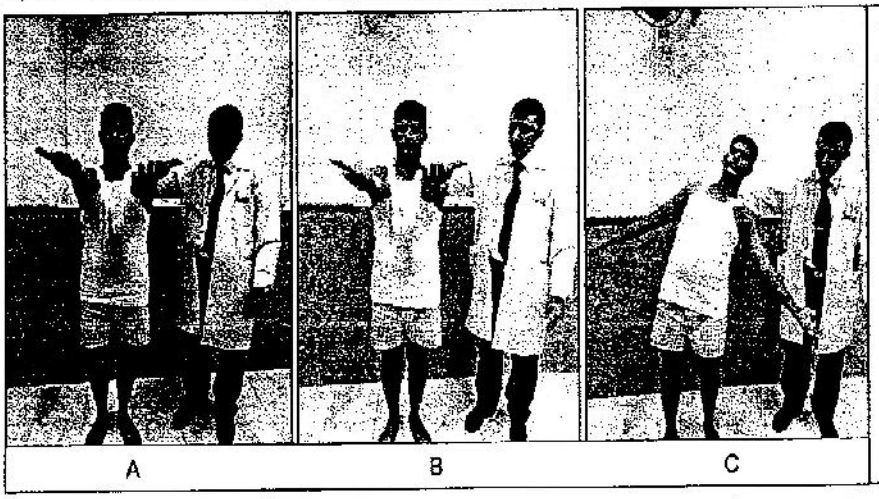


FIGURE 7 – 79. Testing for stance (A) and performing Romberg's test (B) which is shown when it is positive (C).

GAIT EXAMINATION

Make sure that the patient's legs are clearly visible. Now ask the patient (FIGURE 7 – 80) to:

STEP I	Walk normally for a few meters, with a walking aid if needed, then turn around and return
STEP II	Then ask the patient to walk heel-to-toe to exclude a midline cerebellar lesion (tandem gait)
STEP III	Then ask the patient to walk on the toes (an S1 lesion will make this difficult or impossible)
STEP IV	Then ask the patient to then walk on the heels (an L4 or L5 lesion causing foot drop will make this difficult or impossible)
STEP V	Then test for proximal myopathy by asking the patient to squat and then stand up, or sit in a low chair and then stand.

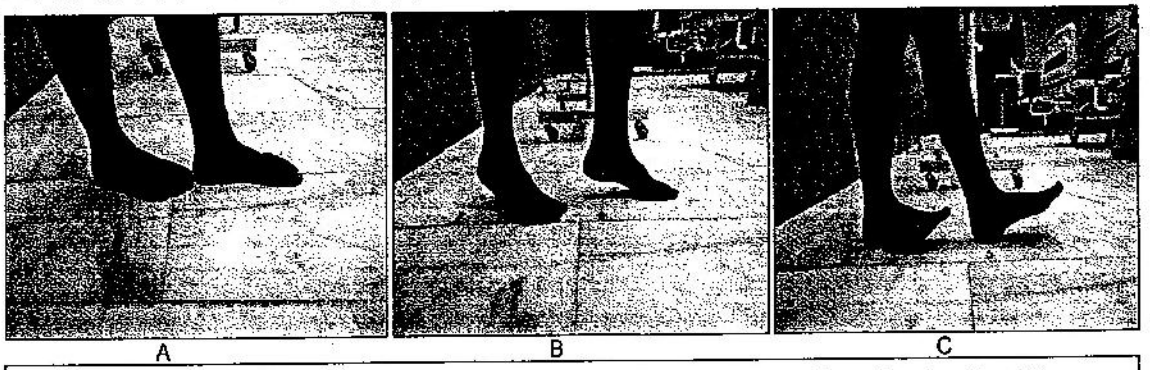


FIGURE 7 – 80. Gait examination should include normal gait and tandem (A), toe (B), and heel walking (C).

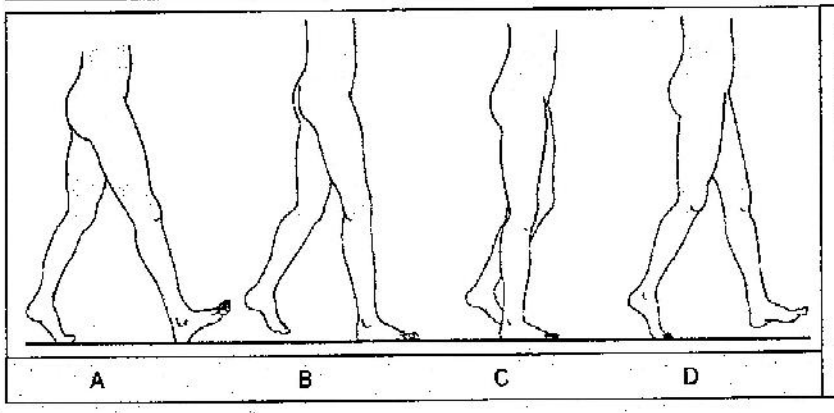


FIGURE 7 – 81. The stance phase of the gait consists of heel-strike (A), foot flat (B), mid-stance (C), and push-off (D).

Normal gait is smooth and symmetrical with each leg 50% out of phase with the other (FIGURE 7 – 81). For each leg, gait has two phases: stance and swing. Stance phase is present when the foot is on the ground and bears weight (50% of the walking cycle) and the swing phase is present when the foot moves forward and does not bear weight (50% of the cycle). Abnormal gait may take one of the following forms (FIGURE 7 – 82):

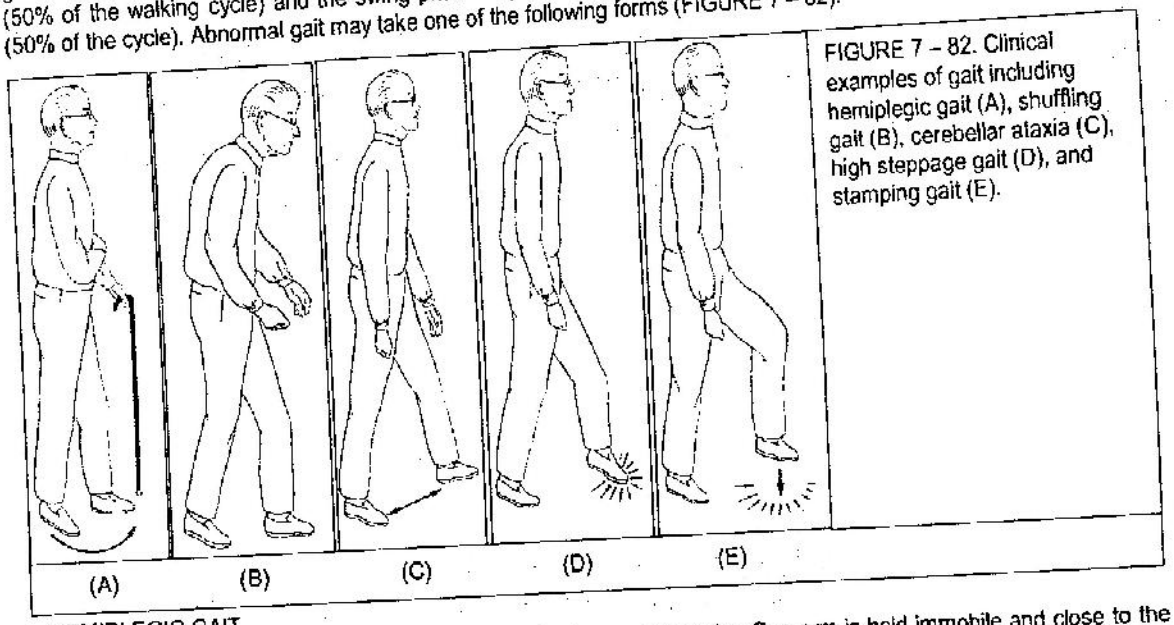


FIGURE 7 – 82. Clinical examples of gait including hemiplegic gait (A), shuffling gait (B), cerebellar ataxia (C), high steppage gait (D), and stamping gait (E).

I. HEMIPLEGIC GAIT

This gait is due to a unilateral upper motor neurone lesion as with stroke. One arm is held immobile and close to the side, with elbow, wrist, and interphalangeal joints flexed. The leg is extended, with plantar flexion of the foot. On walking, the patient either drags the foot, often scraping the toe, or circles it stiffly out-ward and forward (circumduction).

II. SPASTIC GAIT (SCISSORS GAIT)

This gait is associated with bilateral spastic paraperesis of the legs. The gait is stiff and described as toe walking due to spastic calf. Each leg is advanced slowly, and the thighs tend to cross forward on each other at each step (walking through water).

III. HIGH STEPPAGE GAIT (FOOT DROP)

Steppage gait results from an inability to dorsiflex the foot. The patient compensates by exaggerated elevation of the flexed hip and knee to allow the foot to clear the ground while walking. This abnormality is usually the consequence of a peripheral nerve disorder such as a peroneal palsy or other neuropathy, but occasionally it results from a radiculopathy or central lesion. Patient with this kind of gait is unable to walk on their heels. The steppage gait may involve one side or both sides.

IV. SENSORY ATAXIA (STAMPING GAIT)

Loss of joint position sense makes walking unreliable, especially in poor light. The feet tend to be placed on the ground with greater emphasis, presumably in an attempt to increase proprioception input. This results in a 'stamping' gait which results from disorders of the dorsal columns in the spinal cord. Patients with this type of gait watch the ground for guidance while walking. With eyes closed, they cannot stand steadily with feet together (a positive Romberg sign).

V. CEREBELLAR ATAXIA

Patients with cerebellar dysfunction walk with broad-based, unsteady (ataxic) gait 'like a drunken sailor' (cerebellar function is particularly sensitive to alcohol). Less severe degrees of cerebellar ataxia can be detected by asking the patient to walk tandem gait; patients with vermis lesions are unable to do this. Patients with cerebellar dysfunction cannot stand steadily with feet together, whether their eyes are open or closed.

VI. WADDLING GAIT

During walking, alternate placement of the body's weight through each leg requires careful control of the hips by the gluteal muscles. In proximal muscle weakness, usually caused by muscle disease, the hips are not properly fixed by these muscles and trunk movements are exaggerated, producing a rolling or waddling gait.

VII. SHUFFLING GAIT

In this gait, characteristic of parkinsonian, there are small steps, and the patient hardly raises the feet from the ground (freezing). There is often difficulty in initiating walking, but once it begins the patient hurries (festination) and has difficulty stopping. The Parkinsonian patient has stooped posturing with the head and the neck forward and the hips and the knees flexed and seems always to be trying to catch up with the centre of gravity. There is a lack of the normal arm swing. Walking heel to toe will be difficult. A small-stepped shuffling gait is also seen in normal pressure hydrocephalus. The patient here may have gait ignition failure (foot grasp of the floor) or raise and lower the feet in the same place ("egg walking"), this is called gait apraxia. In multi-infarct states (status lacunaire), usually from small

vessel disease, the patient has a shuffling small-stepped gait that is irregular and hesitant with normal arm swing ("marche a petite pas"). This looks different from the festinant gait of severe parkinsonian patients in that it does not have the variable pace and freezing.

VIII. ANTALGIC GAIT

An antalgic gait is one which is altered to reduce pain. Pain in a lower limb is usually aggravated by weight-bearing, so the patient minimizes the time spent in the stance phase on that side. This results in a 'dot-dash' mode of walking.

If the source of pain is in spine, then axial rotatory movements are minimized, resulting in a slow gait with small paces. Patients with hip pain may lean towards the affected side as this decrease the compression force on the hip joint.

IX. HYSTERICAL

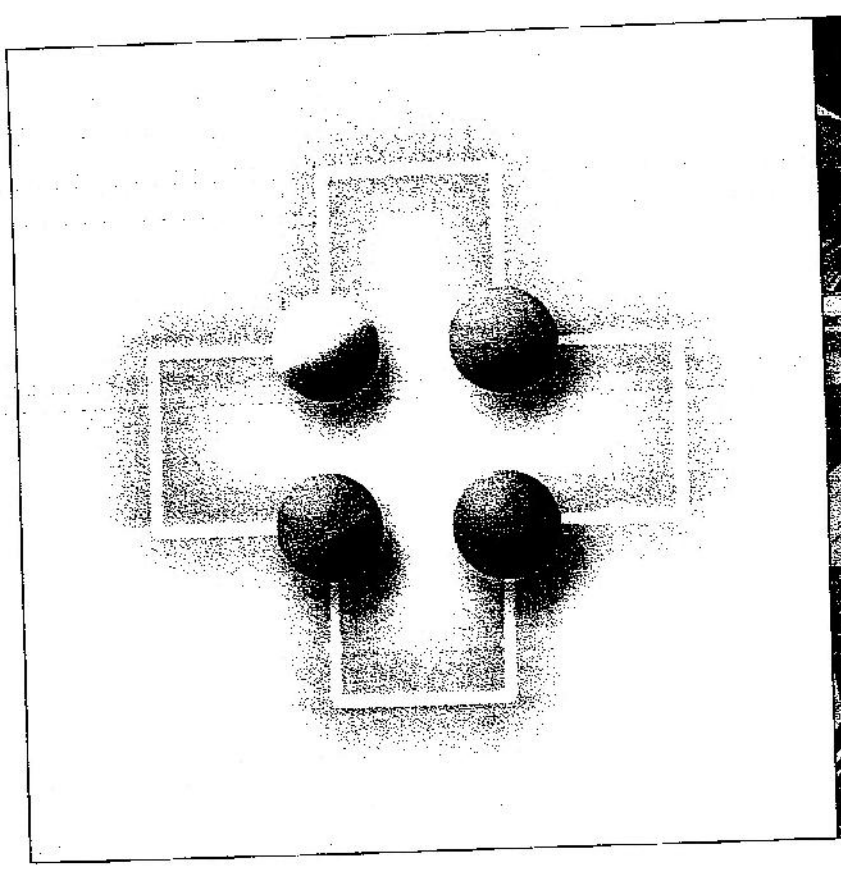
It is also called functional gait. The problems will be variable and inconsistent and no other neurological signs, often with bizarre and elaborate consequences. The patient may fall without causing injury and the gait is often worse when watched.

X. GAIT OF ELDERLY

Speed, balance, and agility decrease with aging. Steps become short, uncertain, and even shuffling. The legs may be flexed at hips and knees. A cane may bolster lost confidence.

8

RHEUMATOLOGIC EXAMINATION



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ANATOMY OF THE MUSCULOSKELETAL SYSTEM

The musculoskeletal system consists of bones, joints, bursae, tendons, ligaments, and skeletal muscles. Bones are divisible into two main types; flat bones e.g. skull and long bones e.g. femur and radius. A thin remnant of cartilage called the growth plate (epiphysis) remains at each end of long bones and is responsible for skeletal growth during childhood and adolescence. Long bones are composed of two types of bone; cortical (compact) bone and canellous (spongy) bone. The cortical bone forms an envelope around the exterior of long bones, enclosing the marrow cavity while the cancellous bone fills the centre of the bone and separated by spaces that are filled with bone marrow. The parts of a long bone are illustrated in FIGURE 8 – 1. Skeletal muscle is an organ, the contraction of which produces movement. Ligaments attach bone to bone, and tendons attach muscle to bone. Cartilage is a type of connective tissue with great resilience. It plays an important role in joint function and in determining bone length. Easing joint action are bursae, roughly disc shaped synovial sacs that allow adjacent muscles or muscles and tendons to glide over each other during movement. The basic functional unit of the musculoskeletal system is the joint. A joint (FIGURE 8 – 1) is a union of two or more bones. There are three primary types of joint articulation (synovial, cartilaginous, and fibrous) allowing varying degrees of joint movement.

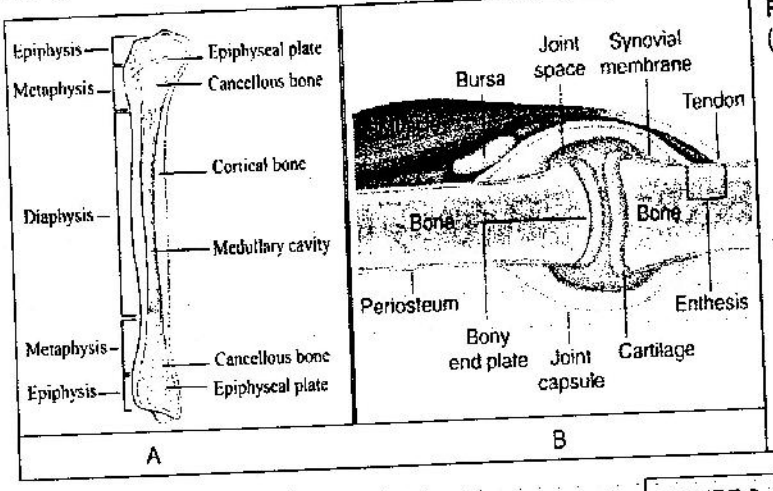


FIGURE 8 – 1. Anatomy of the bone (A) and the joints (B).

ANATOMICAL TERMS

The median plane bisects the body into right and left halves. A plane parallel to the median plane is a sagittal plane. The terms medial and lateral are used in reference to the sagittal plane. A position closer to the median plane is medial; farther from the median plane is lateral. In the upper limb, ulnar is often substituted for medial, and radial is used to denote lateral. In the lower limb, tibial is used to denote medial, and peroneal or fibular is substituted for lateral. The front of the body is the anterior, or ventral, surface, and the back of the body is the posterior, or dorsal, side. The palmar, or volar, aspect of the hand is the anterior surface. The dorsal aspect of the foot faces upward, and the plantar aspect is the sole. Proximal refers to the part of an extremity that is closest to its root; distal refers to the part farthest from the root.

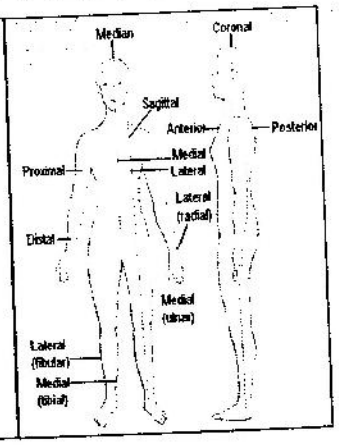


FIGURE 8 – 2. Anatomical terms used in musculoskeletal system.

Joints movements are always described from the neutral position as follows:

1	Flexion	Bending at a joint from the neutral position
2	Extension	Straightening a joint back to the neutral position
3	Hyperextension	Movement beyond the normal neutral position
4	Adduction	Movement toward the midline
5	Abduction	Movement away from the midline
6	Inversion	Turning of the plantar surface of the foot inward
7	Eversion	Turning of the plantar surface of the foot outward
8	Pronation	Rotation so that the palmar surface of the hand is directed downward
9	Supination	Rotation so that the palmar surface of the hand is directed upward

MUSCULOSKELETAL SYSTEM EXAMINATION

The musculoskeletal system examination, like other systemic examination, should include general assessment of the patient and then particular concerns to the individualized joints.

GENERAL EXAMINATION PECULIAR TO THE MUSCULOSKELETAL SYSTEM

As we have learned in the general examination the steps include the general look or the first impression and the regional examination. The general look should include age group, sex, awareness, decubitus, state of comfortability, body built, and dyspnea and tachypnea. The regional examination on the other hand should include examination of the hands, face, neck, lower limbs, and peripheral pulses. These should be interpreted with particular association to the musculoskeletal system (FIGURE 8 – 3).

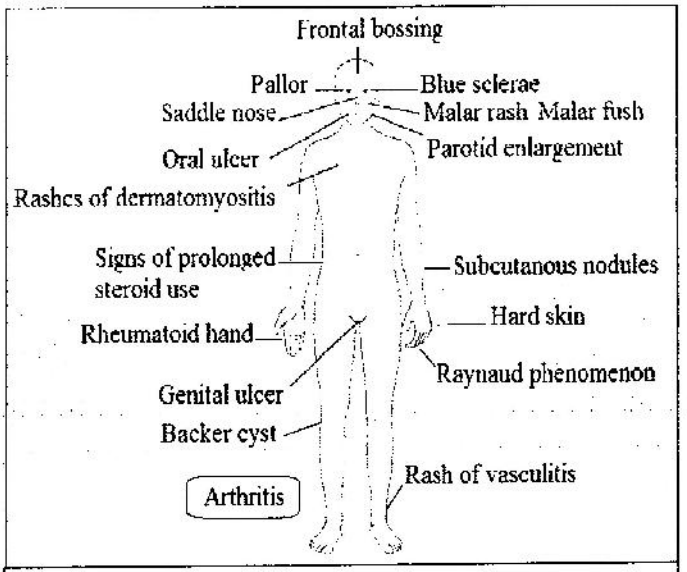


FIGURE 8 – 3. peculiar general signs in the musculoskeletal system.

Frontal bossing refers to the symmetric or asymmetric enlargement of the frontal bone. This abnormality occurs in Paget's disease, acromegaly, rickets disease, and thalassemias. Blue sclerae refer to the bluish discoloration of the both sclerae that occurs in iron deficiency anemia, rheumatoid arthritis, hyperthyroidism, osteogenesis imperfecta, and Ehlers-Danlos syndrome. Mitral flushing occurs in mitral stenosis, carcinoid syndrome, Cushing's syndrome, and due to treatment with niacin or steroid. The erythematous facial (malar) rash with a butterfly distribution across the malar and nasal prominences and sparing of the nasolabial folds is the classic rash of SLE. Similar malar rash may occur in dermatomyositis, erysipelas, and rosacea. Subcutaneous nodules occur in rheumatoid arthritis, osteoarthritis, rheumatic fever, and in chronic tophaceous gout. Subcutaneous nodules in rheumatoid arthritis occur on the extensor surface of the forearm. They are firm and non-tender, and may also be felt at sites of pressure or friction, e.g. the sacrum. Bony nodules in osteoarthritis affect the hand, and are smaller and harder than rheumatoid nodules on the dorsal aspect of the interphalangeal (IP) joints. At the distal interphalangeal (DIP) joints they are called Heberden's nodes, and at the proximal interphalangeal (PIP) joints, Bouchard's nodes. Gouty nodules (tophi) are firm, white, irregular subcutaneous crystal collections. Common sites are the helix of the ear and extensor aspects of the fingers, hands and toes. The overlying skin may ulcerate, discharging crystals, and become secondarily infected. Subcutaneous nodules in rheumatic fever are firm, painless subcutaneous lesions that vary in size from a few millimeters to two cm. The skin overlying them is freely movable and not inflamed. The lesions tend to occur in crops over bone surfaces or prominences and over tendons. Rheumatoid-like hands suggest rheumatoid arthritis, lupus arthritis, psoriatic arthropathy, and osteoarthritis. Saddle nose is a depression of the nasal bridge due to nasal septum destruction and occurs in leprosy, Wagner's granulomatosis, relapsing polychondritis, and congenital syphilis. Hard skin may occur in systemic sclerosis, localized scleroderma (morphea, linear scleroderma, and hemifacial atrophy), overlap syndromes, scleredema and diabetic scleredema, chronic graft-versus-host disease, and paraneoplastic syndrome. While these conditions might be associated with variable skin induration, the distribution pattern of these lesions, absence of Raynaud's phenomenon, and lack of typical visceral organ manifestations or specific autoantibodies help to differentiate these conditions from systemic sclerosis. Backer's cysts are most commonly secondary to rheumatoid arthritis or osteoarthritis. There are a few reported cases secondary to gout and Reiter's syndrome. Raynaud's phenomenon is the abrupt onset of a triphasic color response: well-demarcated pallor of the digits progressing to cyanosis with pain and often numbness followed by reactive hyperemia on rewarming. This vasospastic phenomenon is often precipitated by cold exposure or stress.

Patients with dermatomyositis can present with a variety of photosensitive rashes. Gottron's papules are raised, often scaly, palpable lesions overlying an erythematous base on extensor surfaces such as MCP and PIP joints, elbows, and knees; these are virtually pathognomonic for dermatomyositis. Other characteristic rashes of dermatomyositis are Gottron's sign (macules in a distribution similar to Gottron's papules) and a characteristic purplish rash over and sometimes surrounding the upper eyelids known as the heliotrope rash. Patients may also develop a rash in the V of the neck (V sign) or in the distribution of a shawl (shawl sign). A roughening, scaling, and fissuring of the palmar and lateral aspects of the fingers (mechanic's hands) may be seen in patients with polymyositis or dermatomyositis.

Vasculitic skin rashes may include palpable purpura, livedo reticularis, subcutaneous nodules, ulcers, pulp infarction and digital gangrene.

PRINCIPLE JOINT EXAMINATION

Certain general rules apply to the examination of all the joints and they can be summarized as: look, feel, move, measure, and compare with the opposite side.

LOOK

Remember that joints need to be inspected from the front, the back and the sides. The skin is inspected for erythema indicating underlying inflammation and suggesting active arthritis or infection, bluish discoloration indicating poor blood supply, atrophy suggesting chronic underlying disease, scars indicating previous operations such as tendon repairs or joint replacements, and rashes (psoriasis, SLE, dermatomyositis, and vasculitis). The nails are often also affected. Note any swelling over the joint. Deformity is the sign of a chronic, usually destructive arthritis, and ranges from mild ulnar deviation of the MTP joints in early rheumatoid arthritis to the gross destruction of Charcot's joint. Look for abnormal bone alignment. Subluxation is said to be present when displaced parts of the joint surfaces remain partly in contact. Dislocation is used to describe displacement where there is loss of contact between the joint surfaces. Muscle wasting results from a combination of disuse of the joint, inflammation of the surrounding tissues and sometimes nerve entrapment. It tends to affect muscle groups adjacent to the diseased joint (e.g. quadriceps wasting with active arthritis of the knee) and is a sign of chronicity.

FEEL

Palpate for skin warmth (with the backs of the fingers). A cool joint is unlikely to be involved in an acute inflammatory process. A swollen and warm joint may be affected by active synovitis, infection or crystal arthritis (e.g. gout). Tenderness is a guide to the acuteness of the inflammation or infection. Tenderness can be graded as follows:

Grade I	Patient complains of pain
Grade II	Patient complains of pain and winces
Grade III	Patient complains of pain, winces, and withdraws the joint
Grade IV	Patient does not allow palpation

Palpation of a joint or area for tenderness must be performed gently and the patient's face rather than the joint itself should be watched for signs that the examination is uncomfortable. Palpate the joint for evidence of synovitis which is a soft and spongy (boggy) swelling (soft and non fluctuant). This must be distinguished from an effusion (soft and fluctuant) and from bony swelling (hard, immobile, non fluctuant, and suggests osteophyte formation or subchondral bone thickening).

MOVE

Ask the patient to relax and test passive movements (you do the moving) and then active movements (the patient does the moving) at each joint. The joints may have limited extension (called fixed flexion deformity) or limited flexion (fixed extension deformity). Stability of the joint is important and depends largely on the surrounding ligaments. This is tested by attempting to move the joint gently in abnormal directions. Joint crepitus, grating sensation or noise from the joint, indicates irregularity of the articular surfaces. Coarse crepitus are easily felt and reproducible. They indicate joint damage while fine crepitus are localised and heard with stethoscope. They indicate tendon sheath or bursa problem. Special tests are sometimes adapted to aid further diagnosis.

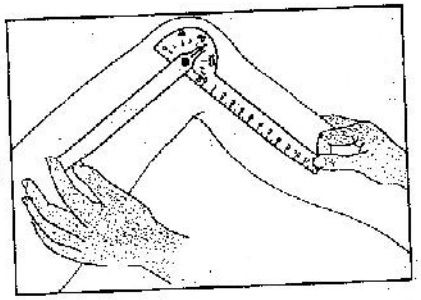
SPECIAL TECHNIQUES

Special techniques are sometimes used to assess the joint function and/or limitation.

CLINICAL CLUE

Measurement of joint movement

Accurate measurement of the range of movement of a joint is possible with a goniometer (FIGURE), which is a hinged rod with a protractor in the centre. The goniometer is not routinely used by non-rheumatologists and there is a wide range of normal values for joint movement. A tape measure is useful for measuring and following serially the quadriceps muscle bulk and in examination of spinal movements.



EXAMINATION OF INDIVIDUAL JOINTS

THE HANDS AND WRIST

LOOK

The wrist includes the distal radius and ulna and eight small carpal bones. The carpal bones lie distal to the wrist joint within each hand. The bones of the hand include five metacarpals, and the proximal, middle, and distal phalanges. Note that the thumb lacks a middle phalanx (FIGURE 8 – 4).

Inspect the wrist joints and the hands for the presence of erythema, atrophy, scars and rashes. Look for swelling and deformity. Then look for muscle wasting of the intrinsic muscles of the hand. This results in the appearance of hollow ridges between the metacarpal bones. Go on to the wrist joints and metacarpophalangeal joints. Look especially for radial deviation of the wrist (deviation of the hand at the wrist joint towards the lateral (radial) side) and ulnar deviation of the fingers (deviation of the phalanges at MTP joints towards the medial (ulnar) side of the hand). These deviations are usually associated with rheumatoid arthritis (FIGURE 8 – 4).

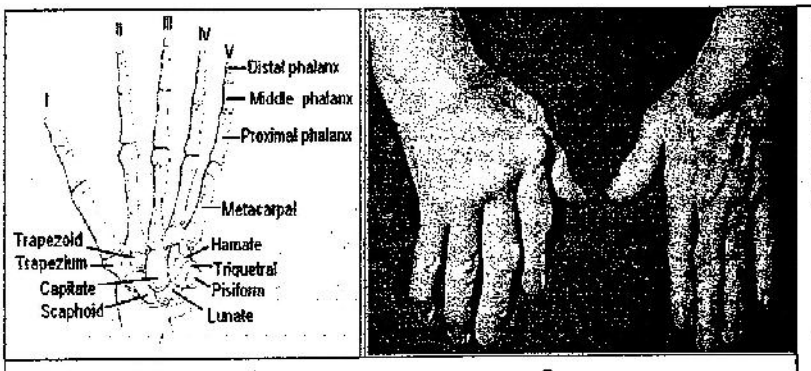


FIGURE 8 – 4. Anatomy of the hand showing the carpal, metacarpal, and phalanges (A). Radial deviation of the wrist and ulnar deviation of the fingers (B) are characteristic for rheumatoid arthritis.

Next inspect the proximal interphalangeal and distal interphalangeal joints. Again note any skin changes and joint swelling. Look for the characteristic deformities of rheumatoid arthritis (FIGURE 8 – 5). These include swan neck and boutonnière deformity of the fingers and Z deformity of the thumb. They are due to joint destruction and tendon dysfunction. The swan neck deformity is hyperextension at the proximal interphalangeal joint and fixed flexion deformity at the distal interphalangeal joint. The boutonnière (buttonhole) deformity consists of fixed flexion of the proximal interphalangeal joint and extension of the distal interphalangeal joints (FIGURE 8 – 5). The Z deformity of the thumb consists of hyperextension of the interphalangeal joint and fixed flexion and subluxation of the MTP joint.

Now look for the characteristic changes of osteoarthritis. The Heberden's nodes are a common deformity caused by marginal osteophytes that lie at DIP joints. Less commonly, the PIP joints may be involved and osteophytes here are called Bouchard's nodes. The only other hand joint to be commonly involved is the carpometacarpal (CMC) joint of the thumb. Patients often complain of pain on use of the thumb, as in wringing out clothes or grasping objects such as screwdrivers and doorknobs. The condition leads to a squared appearance of the base of the hand.

Look also to see if the phalanges appear sausage-shaped. This is characteristic of psoriatic arthropathy, but can also occur in patients with ankylosing spondylitis and Reiter's disease. It is due to interphalangeal arthritis and flexor tendon sheath edema. Finger shortening due to severe destructive arthritis also occurs in psoriatic disease and is called arthritis mutilans (FIGURE 8 – 6). The hand may take up a main en lorgnette ('hand holding long-handled opera glasses') appearance due to a combination of shortening and telescoping of the digits.

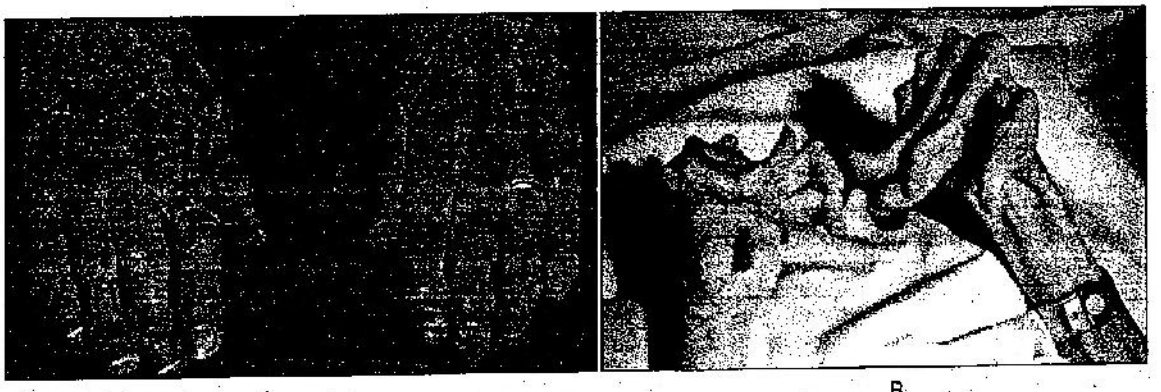


FIGURE 8 – 5. Osteoarhtritis of the hand (A) and rheumatoid arthritis (B).

Characteristic psoriatic nail changes may be visible: these include pitting (small depressions in the nail), horizontal ridging, onycholysis, yellowish discoloration of the nail margins, dystrophic hyperkeratosis, and combinations of these findings. The presence of vasculitic changes around the nail folds implies active disease (FIGURE 8 – 6). These consist of black to brown 1 to 2 mm lesions due to skin infarction and occur typically in rheumatoid arthritis. Splinter hemorrhages may be present in patients with systemic lupus erythematosus, rheumatoid arthritis (and infective endocarditis) and are due to vasculitis. Unlike nail fold infarcts they are located under the nails in the nail beds. Periungual telangiectases occur in systemic lupus erythematosus, scleroderma or dermatomyositis. The hands should now be turned over and the palmar surfaces revealed. Look at the palms for scars (from tendon repairs or transfers), palmar erythema, and muscle wasting of the thenar or hypothenar eminences (due to disuse, vasculitis or peripheral nerve entrapment). Note any thickening of the flexor tendons or flexion contractures in the fingers. Flexion contractures in the little, ring, middle fingers (Dupuytren contractures) arise from thickening of the palmar fascia.

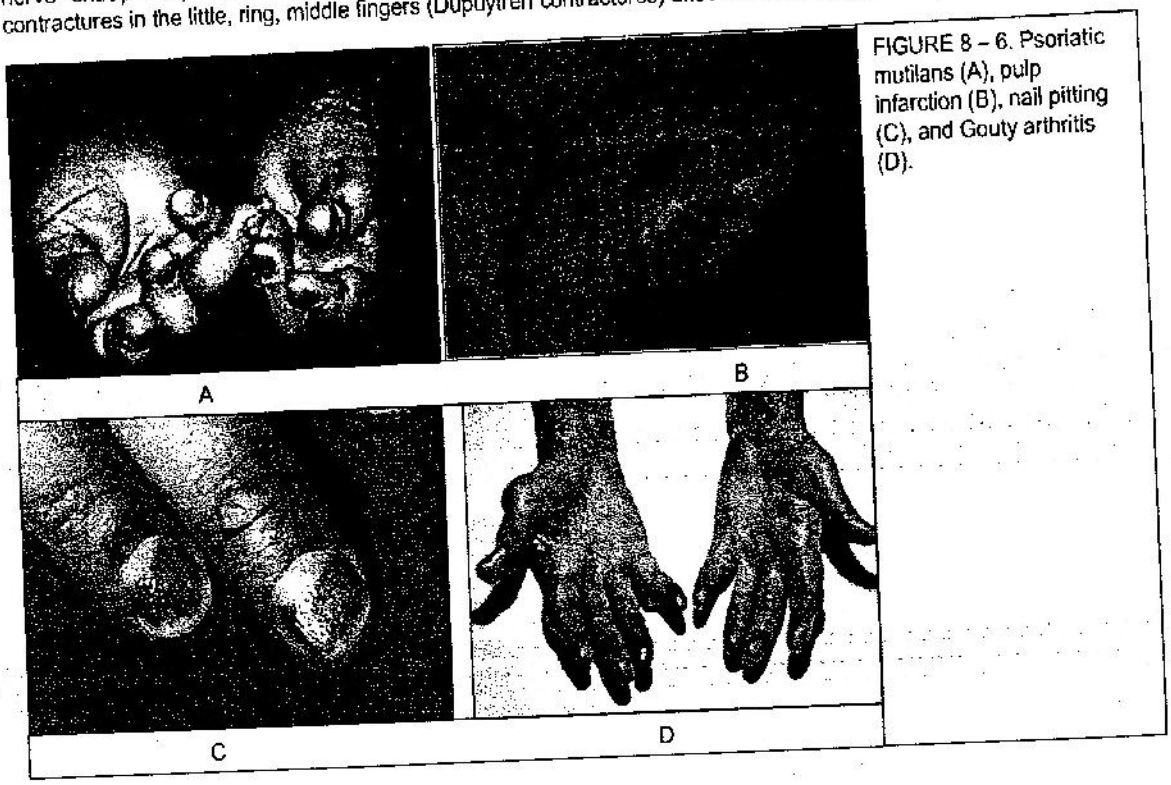


FIGURE 8 – 6. Psoriatic mutilans (A), pulp infarction (B), nail pitting (C), and Gouty arthritis (D).

FEEL

Palpate the wrists with both thumbs placed on the dorsal surface by the wrists supported underneath by the index fingers (FIGURE 8 – 7). Feel gently for synovitis (boggy swelling) and effusions. Palpate the ulnar styloid for tenderness, which can occur in rheumatoid arthritis (FIGURE 8 – 7). Test for tenderness at the tip of the radial styloid. This suggests de Quervain's tenosynovitis. De Quervain's tenosynovitis involves the tendon sheaths of abductor pollicis longus and extensor pollicis brevis, this produces pain maximal over the radial aspect of the distal forearm and wrist. There is tenderness (with or without warmth, linear swelling and fine crepitus) over the distal radius and marked pain on forced ulnar deviation of the wrist with the thumb held in the patient's palm (Finkelstein's sign; FIGURE 8 – 7). It is usually caused by repetitive over-usage, but bilateral symptoms may occur with gonococcal infection.

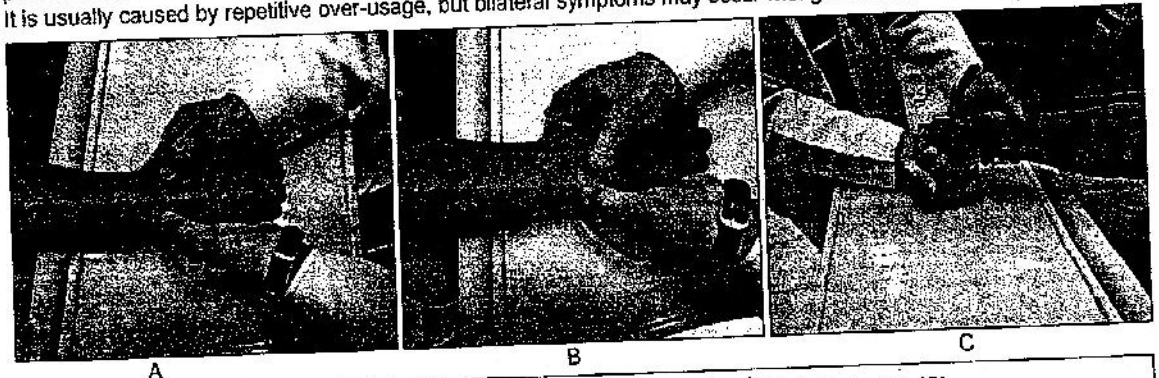


FIGURE 8 – 7. Examination of the wrist (A), ulnar styloid process (B), and Finkelstein's sign (C)

Tenderness over the distal radius occurs in Colles' fracture. Feel for tenderness in the anatomical snuff box if scaphoid injury is suspected. Palpate the carpal bones lying distal to the wrist joint, and then each of the five metacarpals and the proximal, middle, and distal phalanges. Feel for tenderness at the MCP joints by squeezing between the thumb and the index finger (FIGURE 8 – 8). The MCP joints are often boggy or tender in rheumatoid arthritis. Palpate the medial and lateral aspects of each PIP joint between your thumb and index finger, again checking for swelling, bogginess, bony enlargement, or tenderness (FIGURE 8 – 8). PIP changes are seen in rheumatoid arthritis and bony nodes (Bouchard's nodes) occurs in osteoarthritis. Using the same techniques and examine the DIP joints. Bony nodes (Heberden's nodes) on the DIP joints occur in osteoarthritis. Examination of the hands is not complete without feeling for the subcutaneous nodules of rheumatoid arthritis near the elbows.

A B C

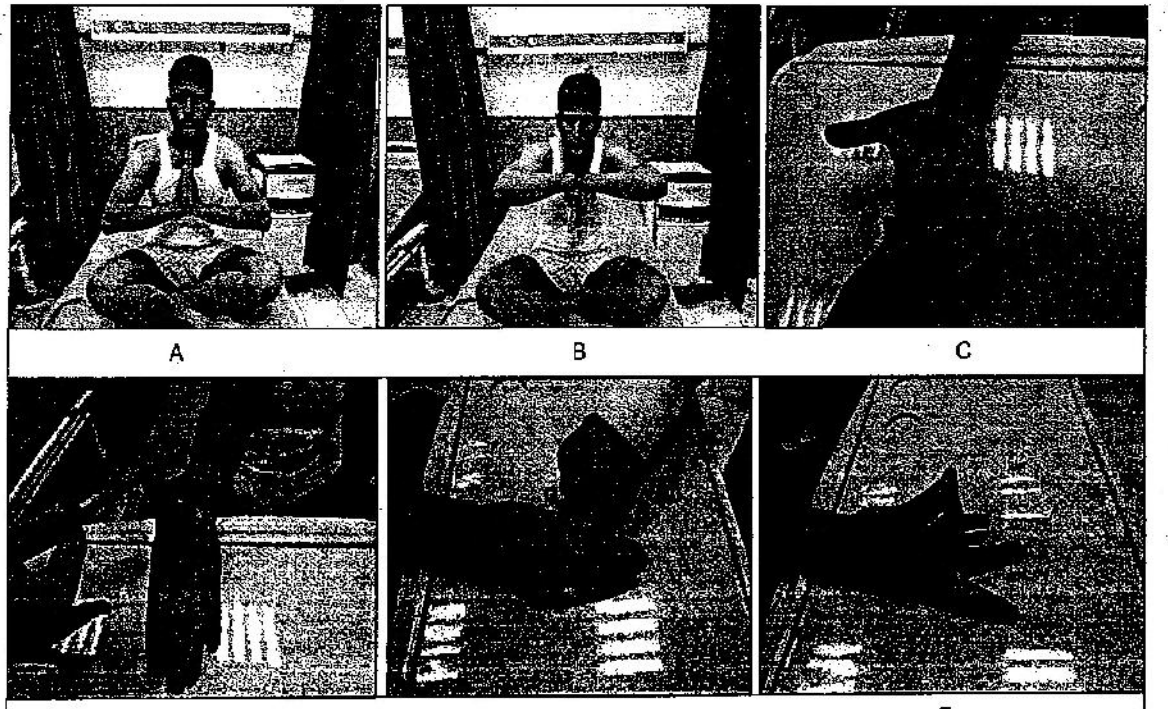
FIGURE 8 – 8. Clinical examination of the anatomical snuff box (A) metacarpophalangeal joints tenderness (B) and the interphalangeal joints (C).

MOVE

Assess active wrist and hand movements before passive movement. Active movements are assessed as follow:

1 Active movements of the wrist joint (FIGURE 8 – 9)

Assess wrist flexion, extension, radial deviation, and ulnar deviation. Compare the two sides



A B C

D E F

FIGURE 8 – 9. Clinical examination of the active wrist extension (A) and wrist flexion (B), active thumb extension (A), active thumb abduction (B), active thumb adduction (C), and active thumb opposition (D).

2 Active movements of the thumb (FIGURE 8 – 9)

The patient holds the hand flat, palm upwards and the examiner's hand holds the patient's fingers. Test extension by asking the patient to stretch the thumb outwards, abduction by asking for the thumb to be pointed straight upwards, adduction by asking him or her to squeeze the examiner's finger, and opposition by getting the patient to touch the little finger with the thumb. Look for limitation of these movements and discomfort caused by them

3 Active movements of the fingers

The metacarpophalangeal and interphalangeal joints movements are tested, as a screening test, by asking the patient to make a fist then to straighten out the fingers (FIGURE 8 – 10). Then test the fingers individually.

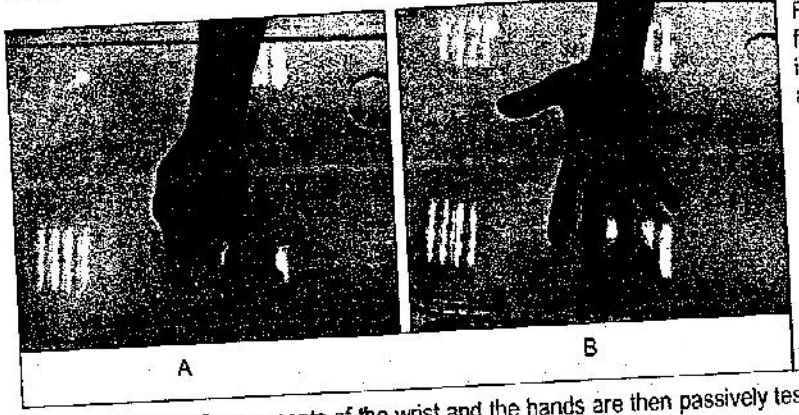


FIGURE 8 – 10. Screening test for MTP joints and interphalangeal joints flexion (A) and extension (B).

The same range of movements of the wrist and the hands are then passively tested. Note any limitation of movement or joint crepitus. Conditions that impair range of motion include arthritis, tenosynovitis, and Dupuytren contracture.

1 The wrist should be passively tested for dorsal flexion (normally 70 degrees) and palmar flexion (also 70 degrees). Then radial and ulnar deviation (20 degrees) is tested
2 Test flexion, extension, abduction, and adduction of the fingers
3 Test thumb flexion, extension, abduction, adduction, and opposition

SPECIAL TECHNIQUES

I. Hand function tests are assessed in the following steps (FIGURE 8 – 11):

1 Hand grip strength is tested by getting the patient to squeeze two of the candidate's fingers
2 Key grip is the grip with which a key is held between the pulps of the thumb and forefinger
3 Opposition strength is where the patient opposes the thumb and individual fingers. The difficulty with which these can be forced apart is assessed
4 Finally, a practical test, such as asking the patient to undo a button or write with a pen, should be performed

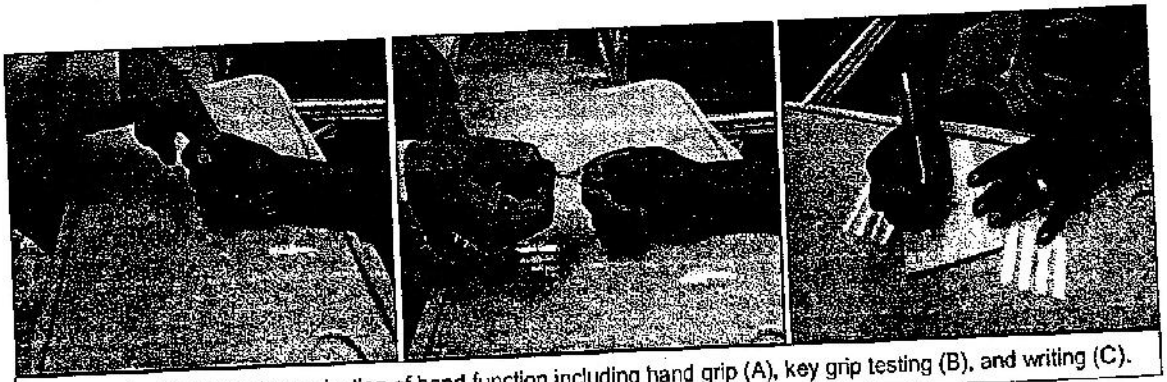


FIGURE 8 – 11. Clinical examination of hand function including hand grip (A), key grip testing (B), and writing (C).

II. Examine then for volar subluxation at the metacarpophalangeal joints (FIGURE 8 – 12)

Volar subluxation can be demonstrated by flexing the metacarpophalangeal joint with the proximal phalanx held between the thumb and forefinger. The metacarpophalangeal joint is then rocked backwards and forwards. Very little movement occurs with this maneuver at a normal joint. Considerable movement may be present when ligament laxity or subluxation is present.

III. Test next for palmar tendon crepitus (FIGURE 8 – 12)
The palmar aspects of the candidate's fingers are placed against the palm of the patient's hand while he or she flexes and extends the metacarpophalangeal joints. Inflamed palmar tendons can be felt creaking in their thickened sheaths and nodules can be palpated. This indicates tenosynovitis. A trigger finger may also be detected by this maneuver. Here the thickening of a section of digital flexor tendon is such that it tends to jam when passing through a narrowed part of its tendon sheath. Rheumatoid arthritis is an important cause. Typically, flexion of the finger occurs freely up to a certain point where it sticks and cannot be extended (as flexors are more powerful than extensors). The application of greater force overcomes the resistance with a snap.
IV. If the carpal tunnel syndrome is suspected, perform Tinel's sign, Phalen's test, and reversed Phalen's test.

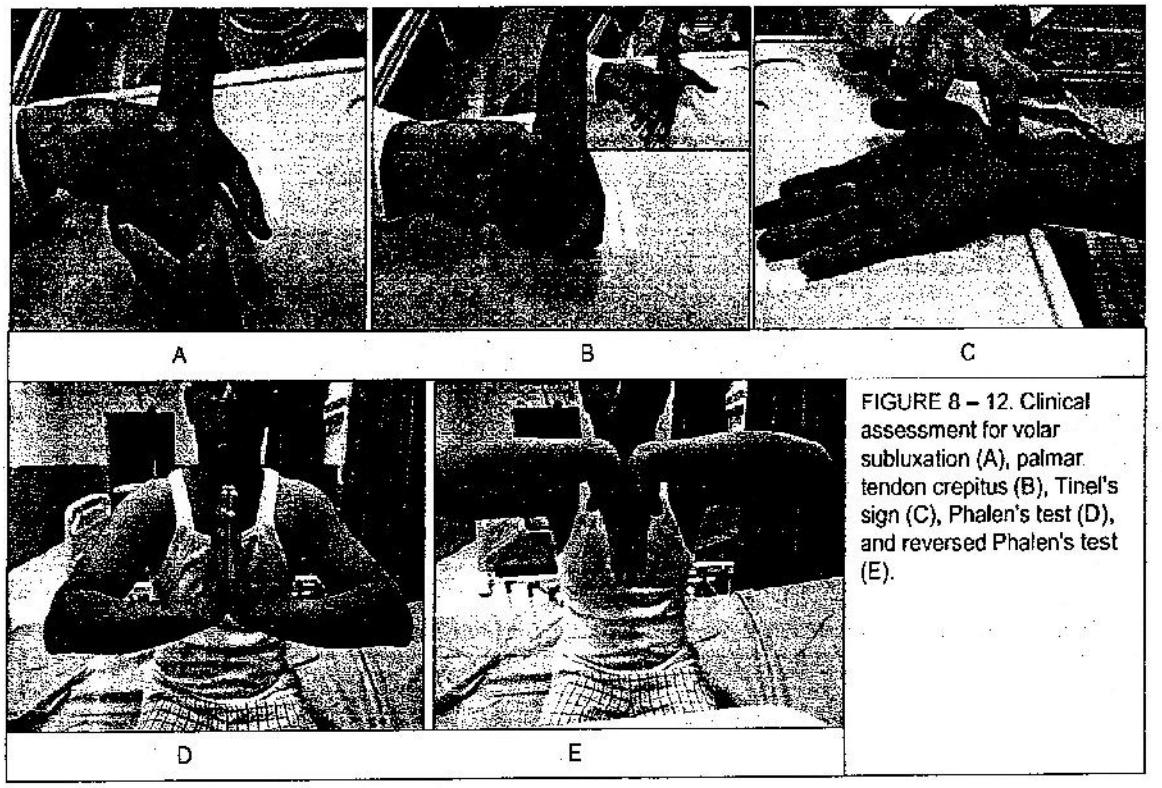


FIGURE 8 – 12. Clinical assessment for volar subluxation (A), palmar tendon crepitus (B), Tinel's sign (C), Phalen's test (D), and reversed Phalen's test (E).

THE ELBOW

The elbow joint is formed by the humerus and the two bones of the forearm, the radius and the ulna. Identify the medial and lateral epicondyles of the humerus and the olecranon process of the ulna. The medial and lateral epicondyles are the flexor and extensor origins respectively for the forearm muscles. These two prominences and the tip of the olecranon are easily palpated. They normally form an equilateral triangle when the elbow is flexed to 90° and lie in a straight line when the elbow is fully extended.

LOOK

The upper arms should be exposed completely. Note any deformity or difference in the normal 5 to 10 degree valgus position (carrying angle) as the patient stands with the palms facing forward. Look for a joint effusion, which appears as a swelling on either side of the olecranon. Discrete swellings over the olecranon or over the proximal subcutaneous border of the ulna may be due to rheumatoid nodules, gouty tophi, or enlarged olecranon bursa.

FEEL

Feel for tenderness, particularly over the lateral and medial epicondyles, which may indicate tennis or golfer's elbow, respectively. Palpate any discrete swellings. Rheumatoid nodules (FIGURE 8 – 13) are quite hard, may be tender and are attached to underlying structures. Gouty tophi have a firm feeling and often appear yellow under the skin, but are sometimes difficult to distinguish from rheumatoid nodules. A fluid collection in the olecranon bursa is softly fluctuant and may be tender if inflammation is present. These collections are associated with rheumatoid arthritis and gout, but often occur independently of these diseases.

MOVE

Assess the active range of movements of the elbow joint and then the passive movements. Elbow movements include flexion, extension, supination, and pronation. The examination technique includes:

1 Assess the extension and flexion (FIGURE 8 – 13)

Ask the patient to touch his shoulder on that side and then straighten the elbow as far as possible. Normal flexion is possible to 150 degrees. Limitation of extension is an early sign of synovitis

2 Assess supination and pronation (FIGURE 8 – 13)

Ask the patient to put his elbows by the side of the body and flex them to 90°. Now ask the patient to turn the hands upwards to face the ceiling (supination: 90°) and then downwards to face the floor (pronation: 85°)

FIGURE 8 – 13. Subcutanous nodules over the extensor surface of the elbow (A), and the clinical techniques for assessing the elbow joint flexion (B) and extension (C); elbow supination (D) and pronation (E).

SPECIAL TECHNIQUES

1 Tennis elbow (lateral epicondylitis) (FIGURE 8 – 14)

Ask the patient to flex the elbow to 90° and pronate and flex the hand/wrist fully. Support the patient's elbow and ask him/her to extend the wrist against your resistance. Pain is produced at the lateral epicondyle and may be referred down the extensor aspect of the arm.

FIGURE 8 – 14. Clinical examination of lateral epicondylitis and medial epicondylitis (B).

2 Golfer's elbow (medial epicondylitis) (FIGURE 8 – 14)

Ask the patient to flex the elbow to 90° and supinate the hand/wrist fully. Support the patient's elbow and ask him/her to flex the wrist against your resistance. Pain is produced at the medial epicondyle and may be referred down the flexor aspect of the arm.

THE SHOULDER

The shoulder joint (FIGURE 8 – 15) consists of the glenohumeral joint and the acromioclavicular joint, but movement also occurs between the scapula and the posterior chest wall (thoracoscapular joints). In the glenohumeral joint, the head of the humerus articulates with the shallow glenoid fossa of the scapula. This joint is deeply situated and not normally palpable and allowing the arm its wide arc of movement; flexion, extension, abduction, adduction, rotation, and circumduction. In the sternoclavicular joint, the convex medial end of the clavicle articulates with the concave hollow in the upper sternum. In the acromioclavicular joint, the lateral end of the clavicle articulates with the acromion process of the scapula.

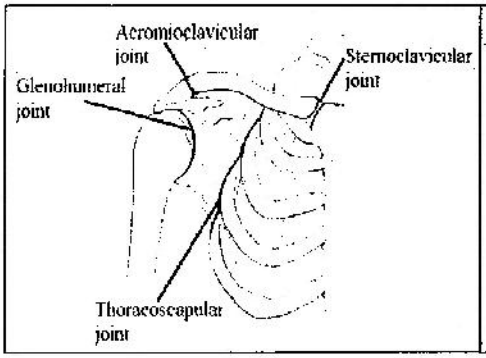
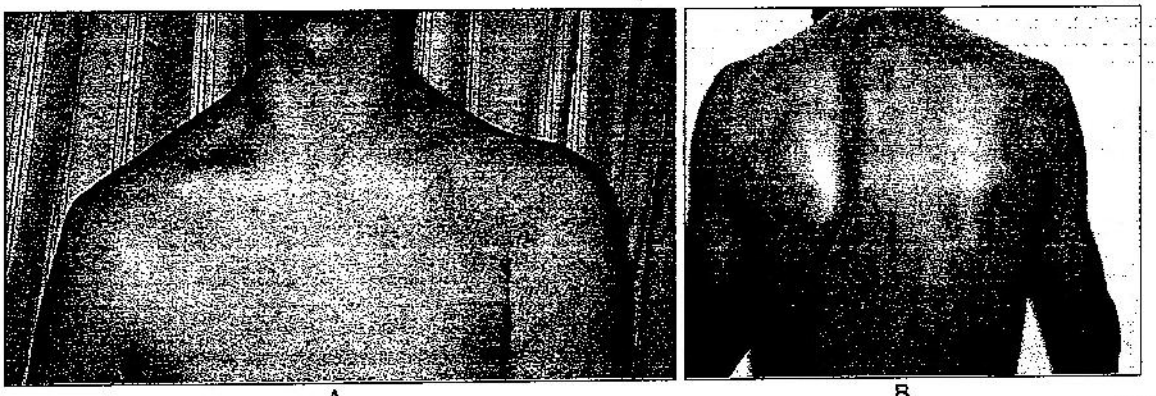


FIGURE 8 – 15. Anatomy of the shoulder joint.

LOOK

Watch the patient undressing and note deformities, swelling, muscle wasting, and the position of the scapula i.e. elevated, depressed or 'winged' (FIGURE 8 – 16). Stand back and compare the two sides. The arms should be held at the same level and the outlines of the acromioclavicular joints should be the same. The deformities of anterior glenohumeral and complete acromioclavicular joint dislocation are obvious (FIGURE 8 – 16). Swelling of the shoulder joint may occur in dislocations, proximal humeral fractures, hemarthrosis and inflammatory conditions. There may be wasting of one of the deltoid muscles that will not be obvious unless the two are compared. Wasting of supraspinatus or infraspinatus indicates a chronic tear of their tendons.



A B

FIGURE 8 – 16. Clinical findings including right anterior glenohumeral dislocation (A) with loss of normal shoulder contour (A) and 'Winging' of the left scapula (B).

FEEL

If there is a history of shoulder pain, ask the patient to point to the painful area. The location of the pain may provide clues as to its origin:

1 Top of the shoulder, radiating toward the neck in acromioclavicular joint problem
2 Lateral aspect of the shoulder, radiating toward the deltoid insertion in rotator cuff problem
3 Anterior shoulder in bicipital tendon problem

Feel for tenderness and swelling starting at the sternoclavicular joint and palpate along the clavicle to the acromioclavicular joint. Palpate the acromion and coracoid (2 cm inferior and medial to the clavicle tip) processes, the scapula spine and the biceps tendon in the bicipital groove.

Palpate the supraspinatus tendon by extending the shoulder to bring supraspinatus anterior to the acromion process. Tenderness is present with ligamentous tears and calcific tendonitis.

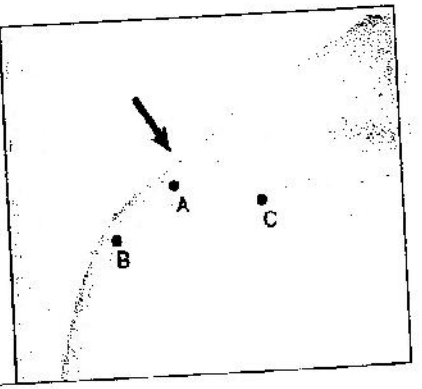


FIGURE 8 – 17. Anatomical landmarks of the shoulder. With your fingers, trace the clavicle laterally. Now, from behind, follow the bony spine of the scapula laterally and up-ward until it becomes the acromion, the summit of the shoulder. Its upper surface is rough and slightly convex. Identify the anterior tip of the acromion (A). With your index finger on top of the acromion, just behind its tip, press medially to find the slightly elevated ridge that marks the distal end of the clavicle at the acromioclavicular joint (shown by the arrow). Move your finger laterally and down a short step to the next bony prominence, the greater tubercle of the humerus (B). Now sweep your finger medially until you feel a large bony prominence, the coracoid process of the scapula (C).

MOVE

During all shoulder movements, limitation with or without pain and joint crepitus are assessed. As a general rule, intra-articular disease produces painful limitation of movement in all directions, while tendonitis produces painful limitation of movement in one plane only, and tendon rupture or neurological lesions produce painless weakness. Test firstly active then passive shoulder movements. Place your hands over the shoulders (FIGURE 8 – 18):

1. Ask the patient to flex and extend the shoulder as far as possible. Flexion is possible to 180 degrees, of which the glenohumeral joint contributes about 90 degrees. With extension, the arm is swung backwards as in marching (65 degrees)
2. To test abduction, ask the patient to lift the arm away from the side of the body. Between 50 and 70% of abduction occurs at the glenohumeral joint (the rest occurs with movement of the scapula on the chest wall), but this increases if the arm is externally rotated. To determine how much movement occurs at the glenohumeral joint, palpate the inferior pole of the scapula between your thumb and index finger to detect scapular rotation. During abduction note the degree and smoothness of scapular movement. If the glenohumeral joint is excessively stiff, then movement of the scapula over the chest wall will predominate. If there is any limitation or pain (painful arc) associated with abduction, test the rotator cuff (discussed later)
3. Measure internal and external rotation with the patient's arm by the side of the body and the elbow flexed at 90°. Internal rotation is assessed as the highest spinous process that the patient can reach with the thumb
4. Assess the ability of the deltoid to abduct against resistance; compare side with side

FIGURE 8 – 18. Test the shoulder movements including shoulder flexion (A), shoulder extension (B), abduction (C), internal rotation (D), and external rotation (E).

SPECIAL TECHNIQUES

1 'Apley scratch test' (FIGURE 7 – 19)

Rapid assessment of shoulder movement is possible using the three-step 'Apley scratch test'. Stand behind and ask the patient to scratch an imaginary itch over the opposite scapula, first by reaching over the opposite shoulder, next by reaching behind the neck and finally by reaching behind the back

A B C

FIGURE 8 – 19. The Apley scratch tests including scratching over the opposite shoulder (A), reaching behind the neck (B), and finally by reaching behind the back (C).

2 The 'apprehension' test (FIGURE 8 – 20)

The anterior stability of the shoulder joint is best assessed by the 'apprehension' test. Stand behind the patient, abduct, extend and externally rotate the shoulder while pushing the head of the humerus forwards with the thumb. The patient will strongly resist this maneuver if there is impending dislocation. There will be a similar response if the arm is adducted and internally rotated and posterior dislocation is about to occur

3 Biceps function

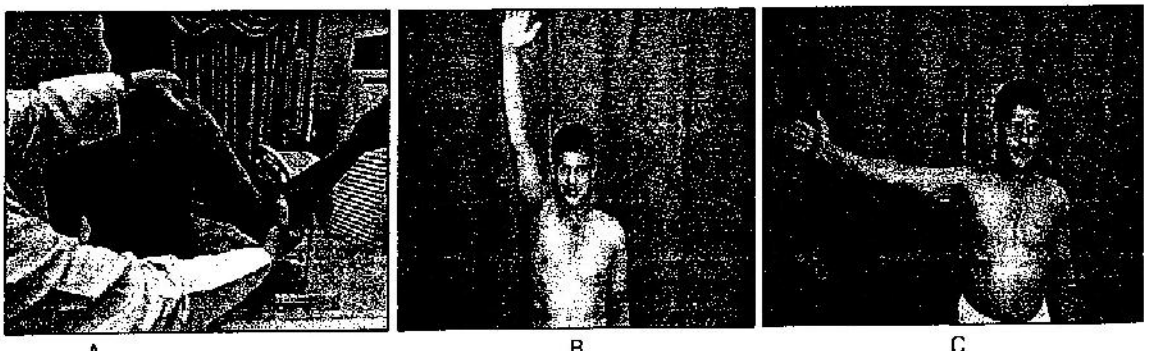
The patient flexes the elbow against resistance. Pain is produced in bicipital tendonitis. A ruptured biceps tendon causes the biceps muscle to roll up into a ball

4 Rotator cuff problems (impingement syndrome/painful arc syndrome) (FIGURE 8 – 20)

The rotator cuff consists of the tendons of the supraspinatus, infraspinatus, subscapularis, and teres minor muscles (SITS). Of the tendons forming the rotator cuff, the supraspinatus tendon is the most often affected, probably because of its repeated impingement (impingement syndrome). Impingement syndrome (painful arc syndrome) occurs in persons participating in baseball, tennis, swimming, or occupations that require repeated elevation of the arm.

Test for a painful arc by passively abducting the arm fully and asking the patient to lower (adduct) it slowly. A painful arc occurs between 60° and 120° of abduction.

If the patient cannot initiate abduction, passively abduct the patient's internally rotated arm to 30-45° while placing your hand over the scapula to confirm there is no scapular movement. Ask the patient to continue to abduct the arm. Pain on active movement, especially against resistance, suggests impingement.



A B C

FIGURE 8 – 20. Apprehension test of the shoulder joint (A) to assess the stability of the shoulder joint and the tests for eliciting painful arc syndrome (B and C) including passive flexion and then active adduction (B) or passive abduction to 30 -45° and then active abduction (C).

THE CERVICAL SPINE

Head nodding occurs at the atlanto-occipital joints, and rotational neck movements mainly at the atlanto-axial joint. Flexion, extension and lateral flexion occur mainly at the midcervical level.

The neural canal contains the spinal cord and the emerging nerve roots, which pass through exit foramina bounded by the facet joints posteriorly and the intervertebral discs and neurocentral joints anteriorly. The nerve roots, particularly in the lower cervical spine, may be compressed or irritated by lateral disc protrusion or by osteophytes arising from the facet or neurocentral joints. A central disc protrusion may press directly on the cord.

LOOK

Ask the patient to remove enough clothing for you to see the neck and upper thorax, then to sit on a chair. Face the patient and observe the posture of the head and neck and note any abnormality or deformity, such as loss of lordosis (usually due to muscle spasm).

FEEL

Feel the midline spinous processes from the occiput to T1 and the paraspinal soft tissues. Note any tenderness or swelling. Feel the supraclavicular fossae for cervical ribs or enlarged cervical lymph nodes.

MOVE

Assess active movements first (FIGURE 8 – 21). Flexion is tested by asking the patient to try to touch his or her chest with the chin (normal flexion is possible to 45 degrees). Extension is tested by asking the patient to look up and back (normally possible to 45 degrees). Lateral flexion is tested by getting the patient to touch his or her shoulder with the ear; lateral bending is normally possible to 45 degrees. Rotation is tested by getting the patient to look over the shoulder to the right and then to the left. This is normally possible to 70 degrees.

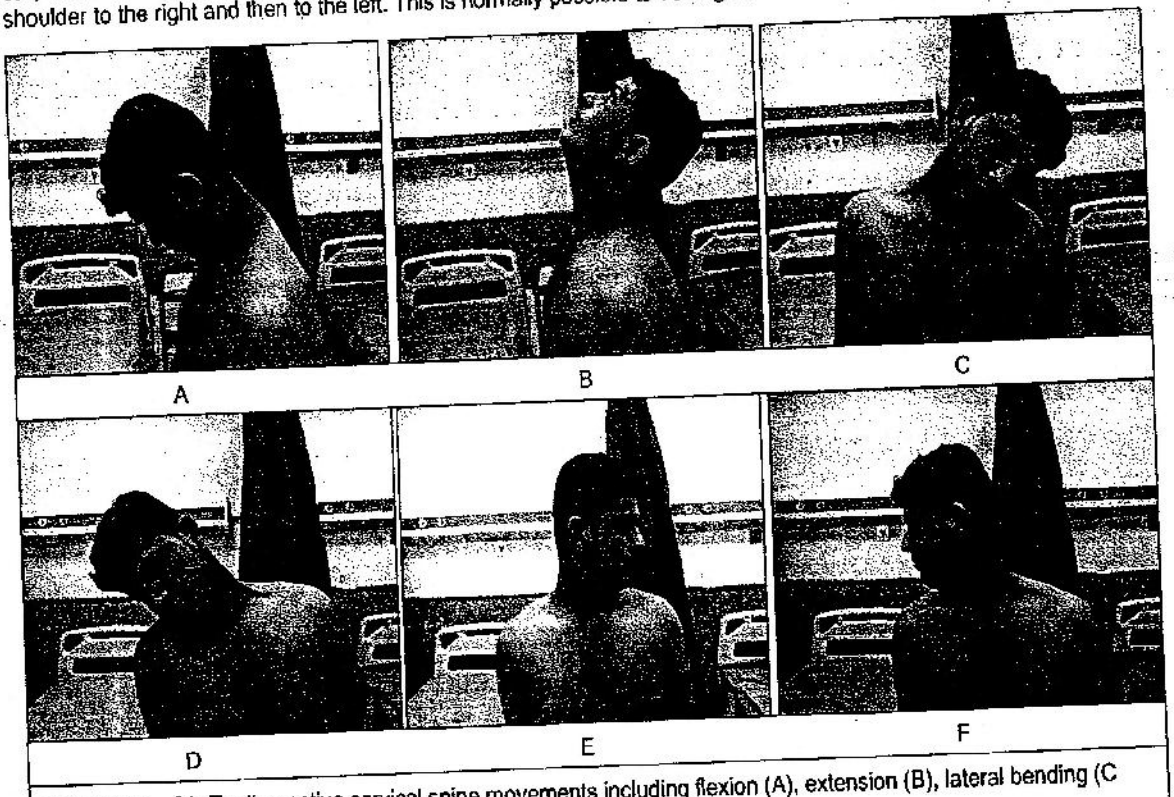


FIGURE 8 – 21. Testing active cervical spine movements including flexion (A), extension (B), lateral bending (C and D), and lateral rotation (E and F).

Gently perform passive movements, if there are reduced active movements, and see if the end of the range has a sudden or a gradual resistance and whether it is pain or stiffness that restricts movement.

THE THORACOLUMBAR SPINE AND SACROILIAC JOINTS

The spine is the central supporting structure of the trunk and back. Note the concave curves of the cervical and lumbar spine and the convex curves of the thoracic and sacrococcygeal spine. These curves help distribute upper body weight to the pelvis and lower extremities and cushion the concussive impact of walking or running.

LOOK

To start the examination, have the patient standing and clothed only in underpants. Look for deformity, inspecting from both the back and side. Note especially loss of the normal thoracic kyphosis and lumbar lordosis, which is typical of ankylosing spondylitis. Also note any evidence of scoliosis, a lateral curvature of the spine which may be simple ('C' shaped) or compound ('S' shaped) and which can result from trauma, developmental abnormalities, vertebral body disease (e.g. rickets, tuberculosis) or muscle abnormality (e.g. polio).

FEEL

Feel the midline spinous processes from T1 to T12 and the paraspinal soft tissues. Feel for increased prominence of one or more posterior spinal processes, implying anterior wedge-shaped collapse of the vertebral body - often related to osteoporosis.

MOVE

Movement is assessed actively. Bending movements largely take place at the lumbar spine, while rotational (twisting) movements occur at the thoracic spine. Range of movement is tested by observation (FIGURE 8 – 22).

1. Flexion is tested by asking the patient to touch the toes with the knees straight. The normal range of flexion is very wide. Many people can reach only halfway down the shins when the knees are kept straight. As the patient bends look at the spine, there is normally a gentle curve along the back from the shoulders to the pelvis. Patients with advanced ankylosing spondylitis have a flat ankylosed spine and all the bending occurs at the hips
2. Test extension by asking the patient to lean backwards. Patients with back pain usually find this less uncomfortable than bending forwards
3. Lateral bending is assessed by getting the patient to slide the right hand down the right leg as far as possible without bending forwards, and then the same for the left side. This movement tends to be restricted early in ankylosing spondylitis
4. Rotation is tested with the patient sitting on a stool (to fix the pelvis) and asking him or her to rotate the head and shoulders as far as possible to each side

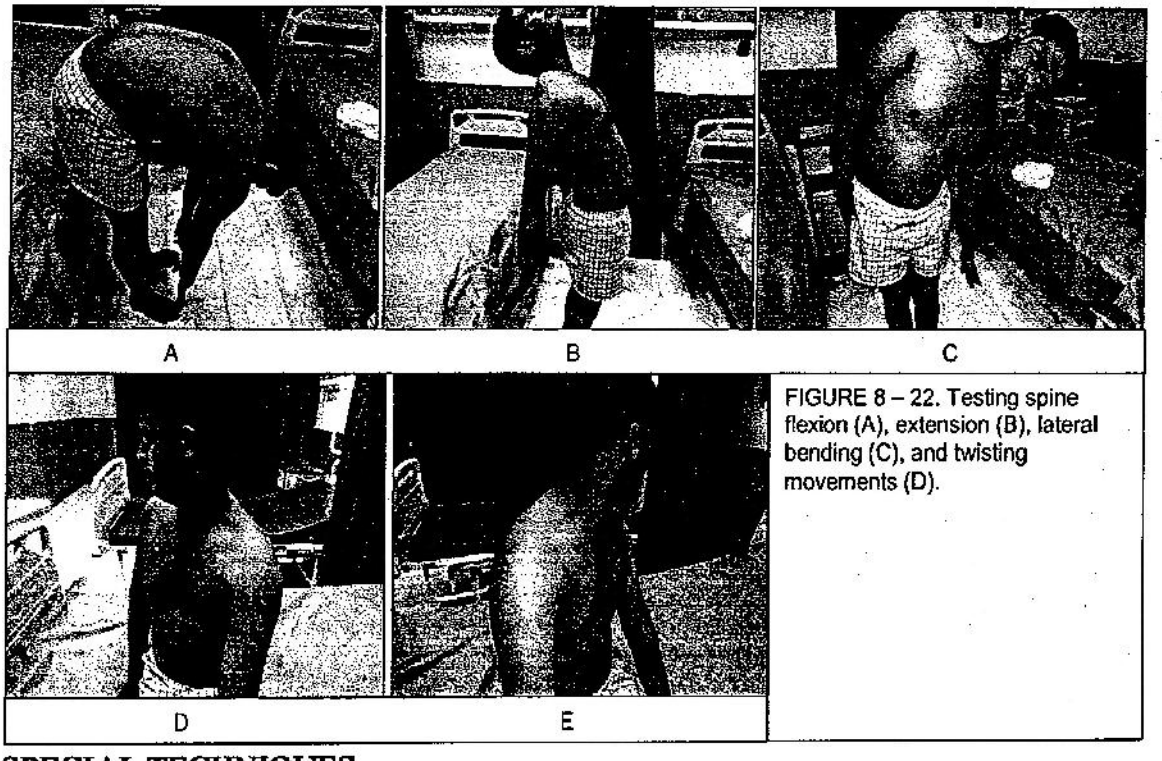


FIGURE 8 – 22. Testing spine flexion (A), extension (B), lateral bending (C), and twisting movements (D).

SPECIAL TECHNIQUES

1. Measure the lumbar flexion with Schober's test (FIGURE 8 – 23). The patient stands erect, with heels together, and marks are made directly over the spine 5 cm below and 10 cm above the lumbosacral junction (identified by a horizontal line between the posterosuperior iliac spines). The patient then bends forward maximally, and the distance between the two marks is measured. The distance between the two marks increases by ≥5 cm in the case of normal mobility and by <4 cm in the case of decreased mobility.

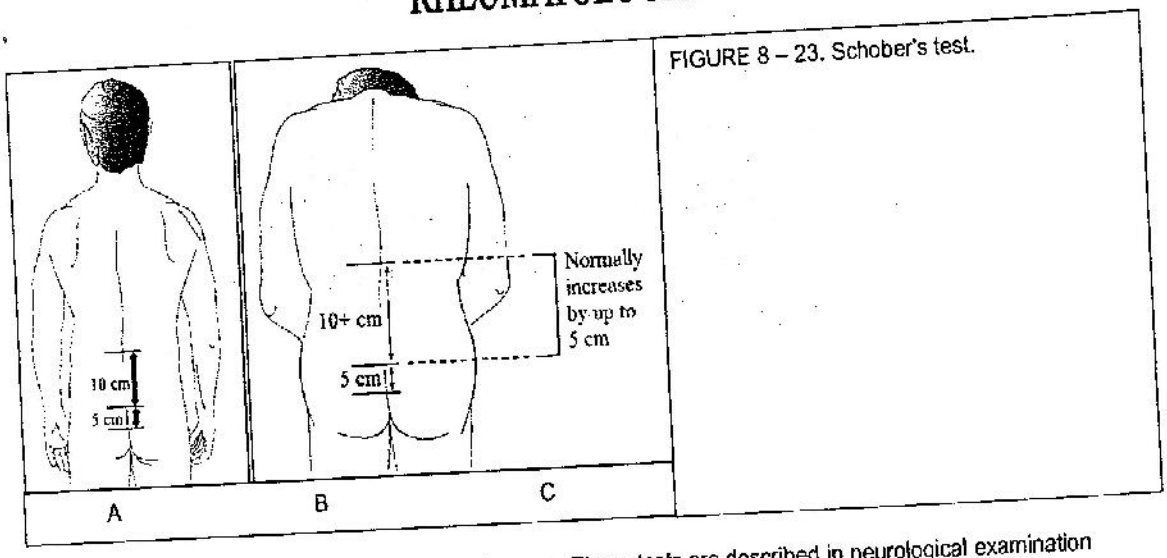


FIGURE 8 – 23. Schober's test.

2 Perform stretch tests for sciatic and femoral nerves. These tests are described in neurological examination

3 The sacroiliac joints lie underneath the dimples of Venus. By tradition, firm palpation with both palms overlying each other is used to elicit tenderness in patients with sacroiliitis. Test each side separately

THE HIP JOINT

The hip is a ball and socket joint and allows movements of flexion, extension, abduction, adduction, internal/external rotation and the combined movement of circumduction. With age, the most common restrictions in movement are extension and internal rotation, followed by abduction.

LOOK

Watch the patient walking and note the use of a walking stick, a slow obviously uncomfortable gait or a limp. Patients should undress to their underwear and remove socks and shoes. Then ask the patient to stand.

1 From the front

Observe whether stance is straight, shoulders are parallel to the ground and placed symmetrically over the pelvis, any pelvic tilt (which may mask a hip deformity or true shortening of one leg), deformities of hip, knee, ankle or foot, and muscle wasting (from polio or disuse secondary to arthritis)

2 From the side

Assess whether there is a stoop or increased lumbar lordosis (both may result from a flexion contracture)

3 From behind

From behind, assess whether the spine is straight or curved laterally (scoliosis); if there is scoliosis, note the relative positions of the shoulders and pelvis, and measure leg lengths. Assess if there is gluteal atrophy

4 Look around the hip and assess whether there are scars, sinuses, dressings or skin changes

FEEL

Feel just distal to the mid-point of the inguinal ligament for joint tenderness. Now feel for the positions of the greater trochanters. The examiner's thumbs are placed on the anterior superior iliac spines on each side while the fore and middle fingers move posteriorly to find the tips of the greater trochanters. These should be at the same level. If one side is higher than the other, the higher side is likely to be the abnormal one. Tenderness on palpation over the greater trochanter suggests trochanteric bursitis.

MOVE

With the patient face up on the couch (FIGURE 8 – 24):

1 Flexion: place your left hand under the back (to detect any masking of hip movement by movement of the pelvis and lumbar spine) and check the range of flexion of each hip in turn (normal 0-120°)

2 Abduction and adduction: stabilize the pelvis by placing your left hand on the opposite iliac crest. With your right hand abduct the extended leg until you feel the pelvis start to tilt (normal 45°). Test adduction similarly by crossing one leg over the other and continuing to move it medially (normal 25°)

3 Internal and external rotation: test with the leg in full extension by rolling the leg on the couch and using the foot to

indicate the range of rotation. Then test with the knee (and hip) flexed at 90°. Move the foot medially to test external rotation and laterally to test internal rotation (normal 45° for each movement)

4 Extension: ask the patient to lie face down on the couch. Place your left hand on the pelvis to detect any movement. Lift each leg in turn to assess the range of extension (normal range 0-20°)

A B C

D

FIGURE 8 – 24. Testing hip joints movements including hip flexion (A), abduction (B) and adduction (C), and extension (D).

SPECIAL TECHNIQUES

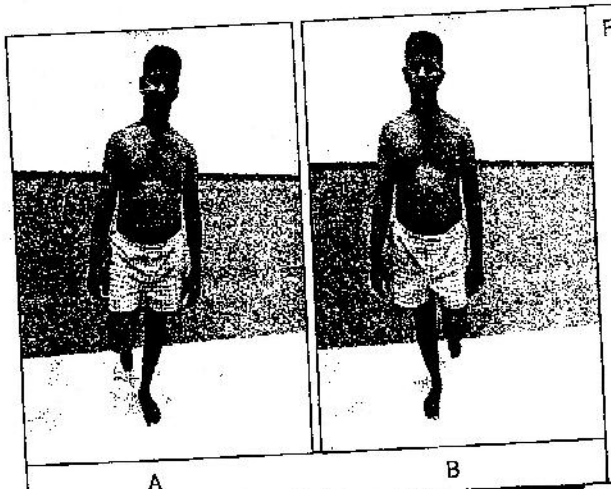
1 Ask the patient to stand and perform the Trendelenburgm test (FIGURE 8 – 25). The patient stands first on one leg and then on the other. Normally the non-weight bearing hip rises, but with proximal myopathy or hip joint disease the non-weight bearing side sags

2 Perform Thomas's test (FIGURE 8 – 26). The patient lies face up on a hard surface. Place your left hand palm upwards under the patient's lumbar spine and passively flex both the patient's legs (hips and knees) as far as possible. Keep the non-test hip maximally flexed (you will feel that the lordotic curve of the lumbar spine remains eliminated). Now ask the patient to extend the test hip. Incomplete extension in this position indicates a fixed flexion deformity at the hip

3 Finally, the true leg length (from the anterior superior iliac spine to the medial malleolus) and apparent leg length (from the umbilicus to the same lower point) for each leg should be measured. A difference in true leg length indicates hip disease on the shorter side, while apparent leg length differences are due to tilting of the pelvis

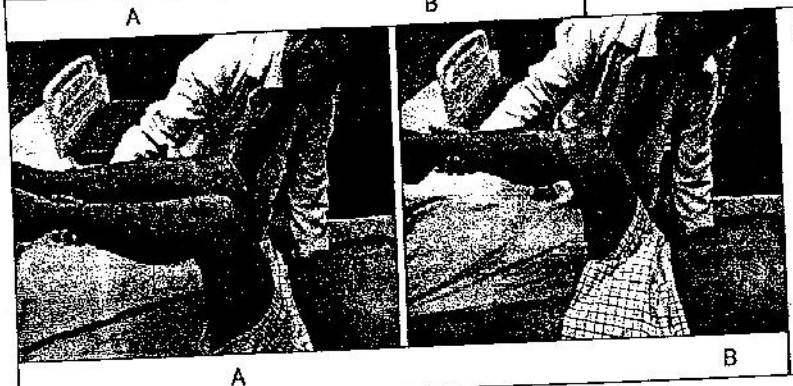
CLINICAL CLUE

In patients with osteoarthritis of the joint, internal rotation, abduction and extension are usually restricted



A B

FIGURE 8 – 25. Trendelenburgm test.



A B

FIGURE 8 – 26. Thomas's test.

THE KNEE JOINT

The knee joint is the largest joint in the body. It involves three bones: the femur, the tibia, and the patella with tibiofemoral and patellofemoral components. It has a synovial capsule that extends under the quadriceps (the suprapatellar pouch), reaching about 5 cm above the superior edge of the patella. The joint is largely subcutaneous, allowing easy palpation of the patella, tibial tuberosity, patellar tendon, and femoral condyles. Flexion is produced by the hamstring muscles. Extension requires the quadriceps muscles, quadriceps tendon, patella, patellar tendon and tibial tuberosity. The medial collateral ligament resists valgus stress. The lateral collateral ligament resists varus stress. The anterior cruciate ligament prevents anterior subluxation of the tibia on the femur, and the posterior cruciate ligament resists posterior translation. The medial and lateral menisci are crescentic fibrocartilaginous structures that lie between the tibial plateaux and the femoral condyles.

LOOK

Look first with the patient lying down on the back with both knees and thighs fully exposed. The affected knee will often be flexed, the most comfortable position. Note any quadriceps wasting which is almost invariable with inflammation or chronic pain and develops within days. Examine the knees themselves for skin changes, scars, swelling and deformity and compare each side with the other. Swelling of the synovium is usually seen medial to the patella and in the joint's suprapatellar extension. Loss of the peripatellar grooves may be an early sign of an effusion. A large effusion forms a horseshoe-shaped swelling above the knee. Look also for Baker's cyst (bursa enlargement in the popliteal fossa). Varus (bow-legs) and valgus deformity (knock-knee) may be obvious here but are more easily seen when patient stands. Varus is often related to osteoarthritis and valgus deformity to rheumatoid arthritis.

FEEL

Feel the quadriceps muscles for wasting. Palpate over the knees for warmth and synovial swelling.
Test carefully for a joint effusion (FIGURE 8 – 27) in the following ways:

1 The patellar tap

The right hand rests over the lower part of the quadriceps muscle and compresses the suprapatellar bursa. The other hand pushes down briskly and firmly over the patella. In a moderate-sized effusion you will feel a tapping sensation as the patella strikes the femur. You may feel a fluid impulse in your left hand.

2 The bulge sign (the ripple test)

The bulge sign is used to detect small effusions. Here the left hand compresses the suprapatellar pouch as for the patellar tap while the fingers of the right hand stroke the medial side of the joint. Now stroke the lateral side of the joint and watch the medial side for a bulge or ripple as fluid re-accumulates on that side.

A B C

D

FIGURE 8 – 27. Testing for patellar effusion including patellar tap and the bulge sign. In patellar tap the candidate must empty the suprapatellar pouch (A) and then press the patella (B) while in bulge sign the candidate also empty the suprapatellar pouch (A), as for the patella tap test and stroke the medial side of the joint to displace excess fluid to the lateral side of the joint (C) and then stroke the lateral side while watching the medial side closely for a bulge or ripple as fluid re-accumulates (D).

MOVE

Move the knee joints actively initially and then passively.

1 Active flexion and extension

With the patient supine ask him to flex the knee up to the chest and then extend the leg back down to lie on the couch (normal range 0-140°)

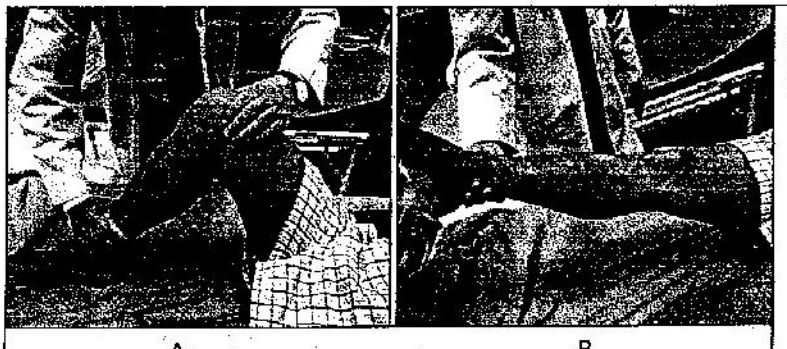
2 Ask the patient to lift the leg with the knee kept straight. If the knee cannot be kept fully extended an extensor lag is present, indicating quadriceps weakness or other abnormality of the extensor apparatus

3 Passive flexion and extension

Test flexion (135 degrees) and extension (5 degrees) by resting one hand on the knee cap while the other moves the leg up and down (FIGURE 8 – 28). The range of movements and the presence of crepitus are noted. Feel for crepitus between the patella and femoral condyles, suggesting chondromalacia patellae (especially in younger female patients) or osteoarthritis. While holding the knee flexed, feel for and attempt to localise tenderness. Feel gently for tenderness along the joint line at the patellar ligament and at the sites of attachment of the collateral ligaments

4 To assess hyperextension (FIGURE 8 – 28)

Lift both legs by the feet. Hyperextension (genu recurvatum) is present if the knee extends beyond the neutral position. Up to 10° is normal



A B

FIGURE 8 – 28. Test for knee flexion (A) and knee hyperextension (B).

SPECIAL TECHNIQUES

1 Lateral and medial collateral ligament (FIGURE 8 – 29)

With the patient's knee fully extended, hold the ankle between your elbow and side. Use both hands to apply a valgus and then varus force to the knee. Movements of more than 5 to 10 degrees are abnormal. Use your thumbs to feel the joint line and assess the degree to which the joint space opens. If the knee is stable, repeat the process with the knee flexed to 30° to assess minor collateral laxity. In this position the cruciate ligaments are not taut

2 The cruciate ligaments (FIGURE 8 – 29)

The candidate steadies the patient's foot by sitting on it. The patient's knee is flexed to 90 degrees. The examiner's hands grasp the tibia and attempt anterior and posterior movements of the leg on the knee joint (anterior drawer sign and posterior drawer sign). Movement may be detected by the examiner's thumbs positioned at the joint margins. Movement of more than 5 to 10 degrees is abnormal. Increased anterior movement suggests anterior cruciate ligamentous laxity and increased posterior movement suggests posterior cruciate ligamentous laxity

The Lachman test may be more accurate. Here the knee is flexed 20 to 30 degrees while the patient is lying supine. Grasp the femur (place your hand above the knee) to steady it, then grab the lower leg below the knee and give it a quick forward tug. It is abnormal when there is exaggerated anterior tibial movement or the knee fails to stop with a thud

3 Patellar stability

Carry out the patellar apprehension test. With the patient's knee fully extended, push the patella laterally and flex the knee slowly. If the patient actively resists flexion, this suggests previous patellar dislocation or instability

4 Meniscal stability (FIGURE 8 – 29)

Ask the patient to lie face up on the couch. Test the medial and lateral menisci in turn:

For the medial meniscus, passively flex the knee to 90 degrees and externally rotate the foot and abduct the upper leg at the hip, keeping the foot towards the midline (i.e. creating a varus stress at the knee) and then extend the knee smoothly. In medial meniscus tears a click or clunk may be felt or heard, accompanied by discomfort

For lateral meniscus, passively flex the knee to 90 degrees and internally rotate the foot and adduct the leg at the hip (i.e. creating a valgus stress at the knee) and then extend the knee smoothly. In tears of the lateral meniscus a click or clunk may be felt or heard, accompanied by discomfort

Lastly perform squat test. Ask the patient to squat, keeping the feet and heels flat on the ground. If he cannot do this, it indicates incomplete knee flexion on the affected side. This may be caused by a tear of the posterior horn of the menisci

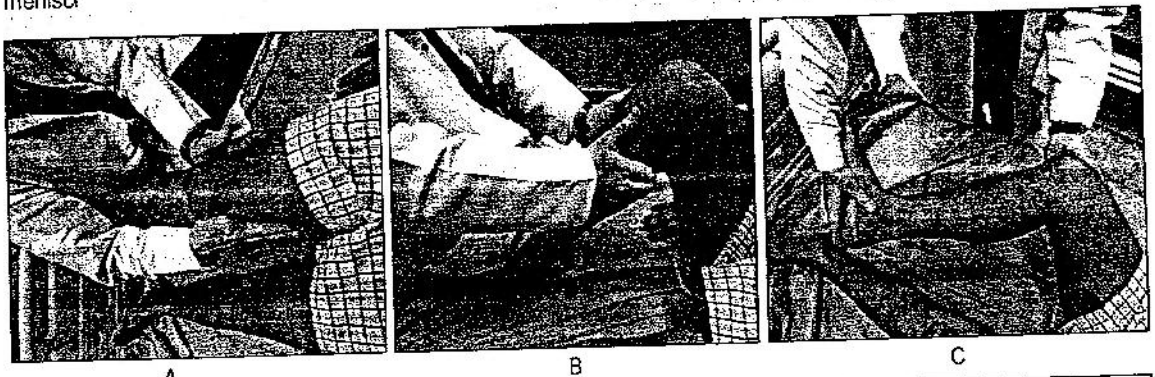


FIGURE 8 – 29. Testing the lateral and medial collateral ligament (A), anterior and posterior cruciate ligaments (B), and meniscal stability (C).

THE ANKLE JOINT AND THE FOOT

The bones can be divided into three regions: the forefoot, midfoot, and hindfoot (FIGURE 8 – 30). The forefoot is represented by the 14 bones of the toes and five metatarsals; the midfoot consists of the three cuneiform bones, the cuboid, and the navicular; the hind foot is composed of the talus and the calcaneus. The foot contains two arches; the longitudinal in the midpart and the transverse in the forepart. The midtarsal joint is formed by the articulations of the navicular to the talus and the calcaneus to the cuboid.

Foot movements are inversion and eversion. Toe movements are dorsiflexion and plantar flexion

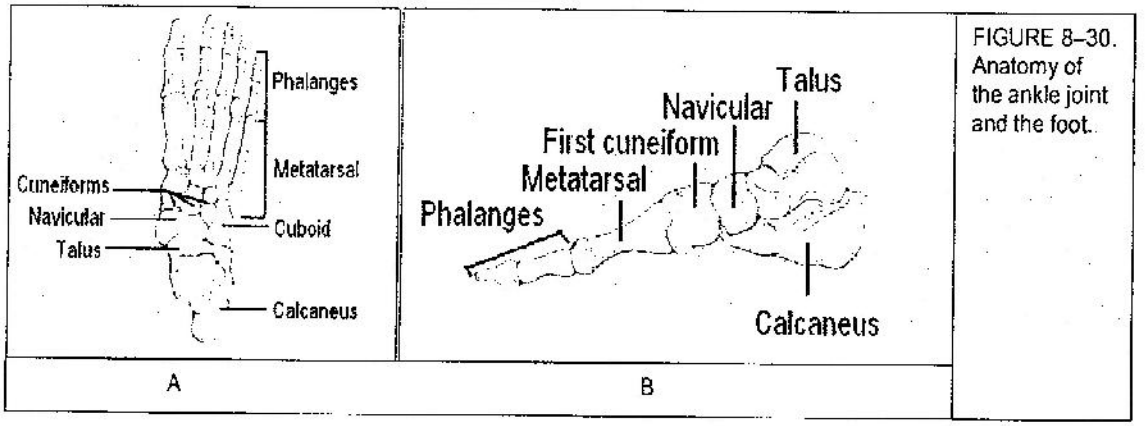


FIGURE 8–30. Anatomy of the ankle joint and the foot.

LOOK

Ask patients to remove their socks and shoes and look for any swelling, scars, deformity or muscle wasting. Deformities affecting the forefoot include hallux valgus (fixed lateral deviation of the main axis of the big toe), clawing (result from dorsiflexion at MTP joints and plantar flexion at PIP and DIP joints), hammer toe (due to dorsiflexion at MTP and DIP joints and plantar flexion at PIP joints) and crowding of the toes, as occurs in rheumatoid arthritis. Sausage deformities of the toes occur with psoriatic arthropathy, ankylosing spondylitis and Reiter's disease. Gout of the first MTP joint causes marked redness and soft tissue swelling. This is followed by desquamation (peeling) of the superficial skin and pain on movement or touch. Swelling of the entire digit, 'sausage toe' (dactylitis), is characteristic of psoriatic arthropathy. Look for the nail changes that suggest psoriasis. Inspect the transverse arch of the foot, which runs underneath the metatarsophalangeal joints, and the longitudinal arch, which runs from the first metatarsophalangeal joint to the heel. These arches, which bear the weight of the body, may be flattened (pes planus - flat foot) or exaggerated (pes cavus). Callosities (an area of thickened skin at a site of repeated pressure) over the metatarsal heads on the plantar surface of the foot occur with subluxation of these joints.

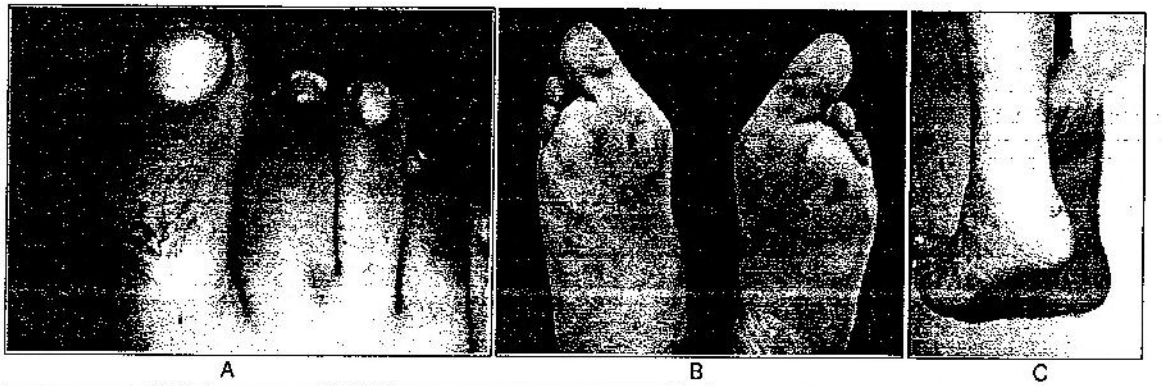


FIGURE 8 – 31. Clinical signs including sausage toe in psoriatic arthropathy (A), rheumatoid feet showing bilateral hallux valgus and calluses over the metatarsal heads (B), and rheumatoid nodule of the Achilles tendon (C).

FEEL

Start from the ankle and feel for swelling around the lateral and medial malleoli. This should not be confused with pitting oedema. Squeeze the metatarsophalangeal joints by compressing the first and fifth metatarsals between your thumb and forefinger. Tenderness suggests inflammation, common in early rheumatoid arthritis. Press upwards from the sole of the foot just proximal to the proximal metatarsophalangeal joints of the third and fourth toes. Pain here suggests Morton's metatarsalgia. This is due to entrapment and swelling of the digital nerve between the toes. It is associated with pain and numbness of the sides of these toes.

Each individual interphalangeal joint is then assessed by feeling. These are typically affected in the seronegative spondyloarthropathies. Extremely tender involvement of the first MTP joint is characteristic of acute gout. In this case the joint also looks red and swollen. Palpate the Achilles tendon for rheumatoid nodules (FIGURE 8 – 31) and tenderness due to Achilles tendonitis.

MOVE

Actively assess the plantar flexion and dorsiflexion at the ankle and inversion/eversion of the foot by asking the patient to perform these movements. Move then the ankle joint, grasping the midfoot with one hand. Dorsiflexion is tested by

raising the foot towards the knee (normally possible to 20 degrees) and plantar flexion by performing the opposite maneuver (normally possible to 50 degrees). With the subtalar joint, only inversion and eversion of the foot on the ankle are tested. Tenderness on movement is more important than range at this joint. The midtarsal (midfoot) joint allows rotation of the forefoot when the hindfoot is fixed. This is done by steadying the ankle with one hand and twisting the forefoot. Again, tenderness rather than range of movement is noted.

SPECIAL TECHNIQUES

Achilles tendon stability (FIGURE 8 – 32)

Ask the patient to kneel with both knees on a chair

Palpate the Achilles tendon for focal tenderness and soft tissue swelling. Achilles tendon rupture is often palpable as a discrete gap in the tendon about 5 cm above the calcaneal insertion

Squeeze the calf and watch for plantar flexion at the ankle. Absence of plantar flexion is a positive Simmond's test indicating rupture of the Achilles tendon. Sudden severe pain "like a gun-shot wound," an ecchymoses from the calf into the heel, and a flat-footed gait with absence of (toe-off) may also be present

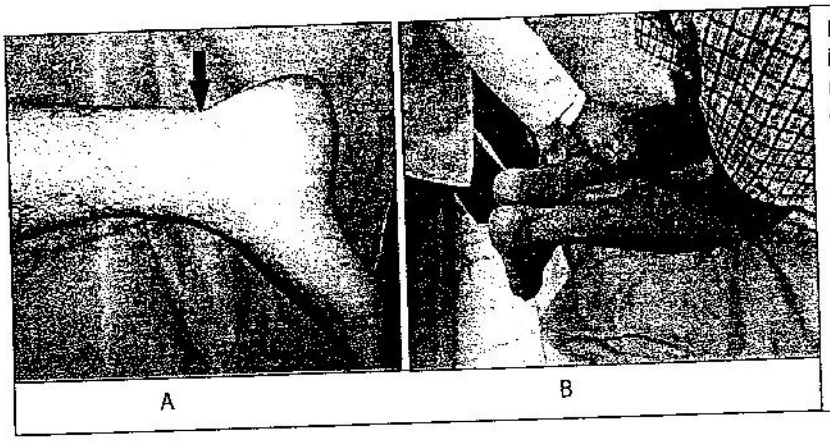
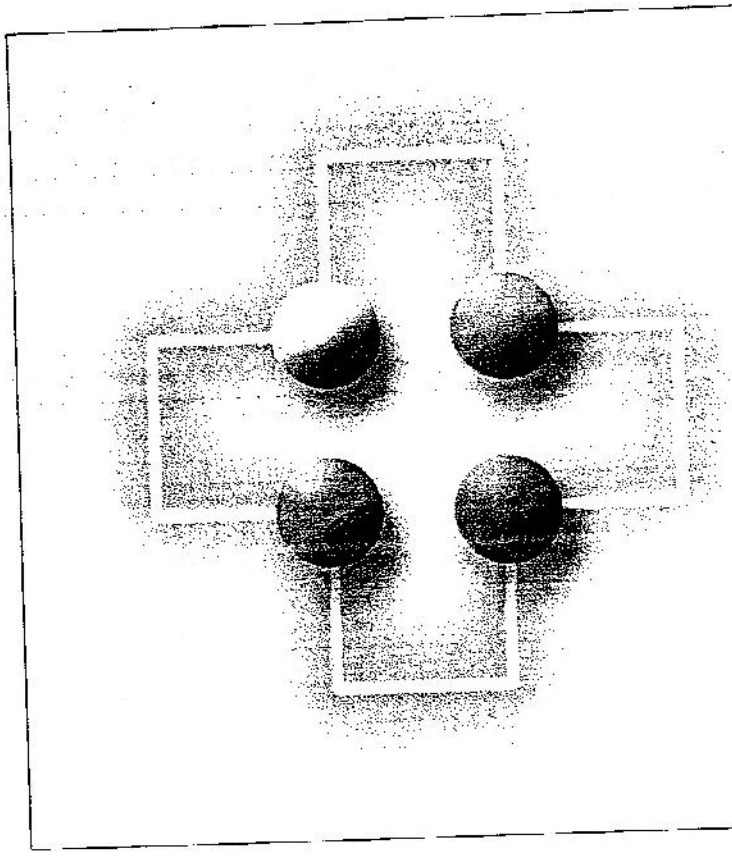


FIGURE 8–32. Focal gap indicating Achilles tendon rupture (A) and the technique of Simmond's test (B).

Further reading

- Hutchison's clinical methods
- Davidson's principles and practice of medicine
- Harrison's principles of internal medicine
- Cecil principles of internal medicine
- Macleod's clinical examination
- Oxford Handbook of Clinical Examination – Practical Skills
- First Aid for the Internal Medicine Boards
- Clinical Examination - A Systematic Guide to Physical Diagnosis
- Textbook of Clinical Neurology
- Queen Square Textbook of Neurology
- Dictionary of Neurological Signs
- Clinical Neurology (McGraw-Hill, 2009)
- 2005 Adams and Victor's Principles of Neurology
- Baily and Love's short practice of surgery
- Gynecology by ten teachers
- Obstetrics by ten teachers
- Hamilton Baily's physical signs in clinical surgery
- Browse; signs and symptoms of surgical diseases
- Nelson's text book of pediatrics
- Bate's guide to physical diagnosis.
- Textbook of physical diagnosis.
- Oxford handbook of clinical examination/ practical skills.
- Kochar's Clinical Medicine for Students.

SPECIFIC EXAMINATIONS



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SPECIFIC EXAMINATIONS

This chapter deals with the following examinations:

1 Thyroid status examination
2 Breast examination
3 Examination of hernia
4 Lump (swelling) examination
5 Ulcer examination
6 Examination of the arteries, veins, and lymphatics

THYROID STATUS EXAMINATION

The thyroid gland envelops the upper trachea and consists of two lobes connected by an isthmus. The normal thyroid is 12–20 g in size, highly vascular, and soft in consistency. As seen from the front, the thyroid is butterfly shaped and wraps around the anterior and lateral portions of the larynx and trachea (FIGURE 9 – 1). The thyroid isthmus lies across the trachea just below the cricoid cartilage of the larynx. The lateral lobes extend along either side of the larynx, reaching the level of the middle of the thyroid cartilage of the larynx. Occasionally, the thyroid gland may extend downward and enlarge within the thorax, producing a substernal goiter. Goiter (BOX 9 – 1) is a structural term meaning generalized enlargement of the thyroid gland. A patient with goiter may have euthyroid state, hyperthyroidism or hypothyroidism. Euthyroid means a normal level of thyroid hormones; hypothyroidism means decreased level of thyroid hormone; hyperthyroidism means increased level of thyroid hormone. Thyrotoxicosis is defined as the state of thyroid hormone excess and is not synonymous with hyperthyroidism, which is the result of an excessive thyroid function. Thyrotoxicosis is divided into four stages:

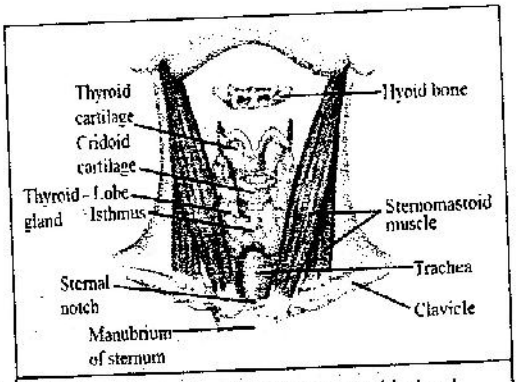


FIGURE 9 – 1. Structure of the thyroid gland

1 Mild thyrotoxicosis

Mild thyrotoxicosis consists of lid retraction and lid lag. Lid retraction means widening of the palpebral fissure due to sympathetic over-activity consequently lead to contraction of the striated fibers of the levator palpebrae superioris. Lid lag means the upper eyelid seems to lag behind the eye ball when the patient looks downwards

2 Moderate thyrotoxicosis

This state includes exophthalmos which is bilateral forward displacement of the eye ball. When it is unilateral, it is called proptosis. This exophthalmos is due almost entirely to increase deposition of fat

3 Severe thyrotoxicosis

The increased deposition of fat (intraorbital fat) is superimposed by intraorbital edema and cellular infiltration of mucous membrane as well as involvement of oculomotor nerve. It is characterised by watering of the eyes, conjunctival congestion, increased intraobital pressure, ophthalmoplegia (double vision due to edema)

4 Progressive exophthalmos

This state supervenes on the severe case in otherwise successful treatment. The exophthalmos continues to increase instead of regress and the visual acuity becomes impaired. This state is characterized by chemosis, impairment of the corneal sensitivity, and exophthalmic ophthalmoplegia

Thyroid status examination consists of two parts; general examination and the thyroid gland examination.

GENERAL EXAMINATION PECULIAR TO THE THYROID GLANDS

As we have learned in the general examination the steps include the general look or the first impression and the regional examination. The general look should include age group, sex, awareness, decubitus, state of comfortability, body built, and dyspnea and tachypnea. The regional examination on the other hand should include examination of the hands, face, neck, lower limbs, and peripheral pulses. These should be interpreted with particular association to the thyroid gland. Particular concerns should be paid toward the eye and thyroid gland examination. The candidate should follow the steps provided below.

BOX 9 – 1. Causes of thyroid enlargement

Diffuse goiter		Multinodular goiter	Solitary nodule
Dyshormonogenesis[1] Iodine deficiency[1] (endemic goiter) Simple goiter[1] Hashimoto's thyroiditis[1] Suppurative thyroiditis[2] Transient thyroiditis[2] Riedel's thyroiditis[2]	Graves' disease Drugs (iodine, amiodarone, lithium) Infiltrative (amyloidosis, sarcoidosis)	Multiple nodules that represent multiple adenomas or focal hyperplasia	Simple cyst Colloid nodule Follicular adenoma Papillary carcinoma Follicular carcinoma Medullary cell carcinoma Anaplastic carcinoma Lymphoma Metastasis

[1] Goiter is likely to shrink with thyroxin therapy (DISH)
[2] Usually tender goiter

EYE EXAMINATION DURING THYROID STATUS EXAMINATION

The relationship of the eye to the limbus (FIGURE 9 – 2) is shown below:

I Normal eye when the upper eye lid is midway between the pupil and the superior part of limbus. Lower eye lid is at the inferior part of limbus

II Lid retraction when the upper eye lid is raised while the lower eyelid is still normally in its site

III Exophthalmos means both eye lids moved away from the center with the sclera visible below or all around the cornea. The eyeballs will be bulged forward.

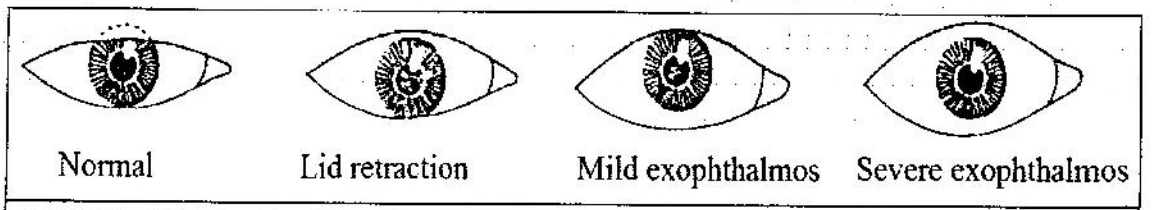


FIGURE 9 – 2. Relationship of the eyelids with the cornea

The sequence of examination should include the following (FIGURE 9 – 3):

1 Inspect the eyes from the front

Detect lid retraction (Stellwag's sign)

Detect exophthalmos

2 Inspect the eyes from the side

Detect any forward bulging that is indicative of proptosis

Detect lid lag (von Graefe's sign)

The patient is asked to follow the candidate's finger moves up and down ward slowly several times without being moves her head. Any lag in upper eye lid means a positive sign

3 Perform Naffziger's method

Stand behind the seated patient and tilt her head back ward so the plane of vision will be from your eye to the superciliary ridges to the cheek of the patient

4 Return back to inspect the eyes from the front and

Ask the patient to bend her head down and look upward. Absence of wrinkling indicates exophthalmos (Joffroy's sign)

Ask the patient to move her eyes laterally while her head is fixed to see the dilated blood vessels and watering of the eyes as well as conjunctival congestion (chemosis), and corneal ulceration (due to inability to close the eyelids)

Light pressure is applied on the globes when the eyes are closed to detect increased resistance more than normal

Perform H – test to detect limitation of movement and nystagmus and to ask about diplopia.

Perform near accommodation reflex. Difficulty in convergence indicates exophthalmos (Moebius sign)

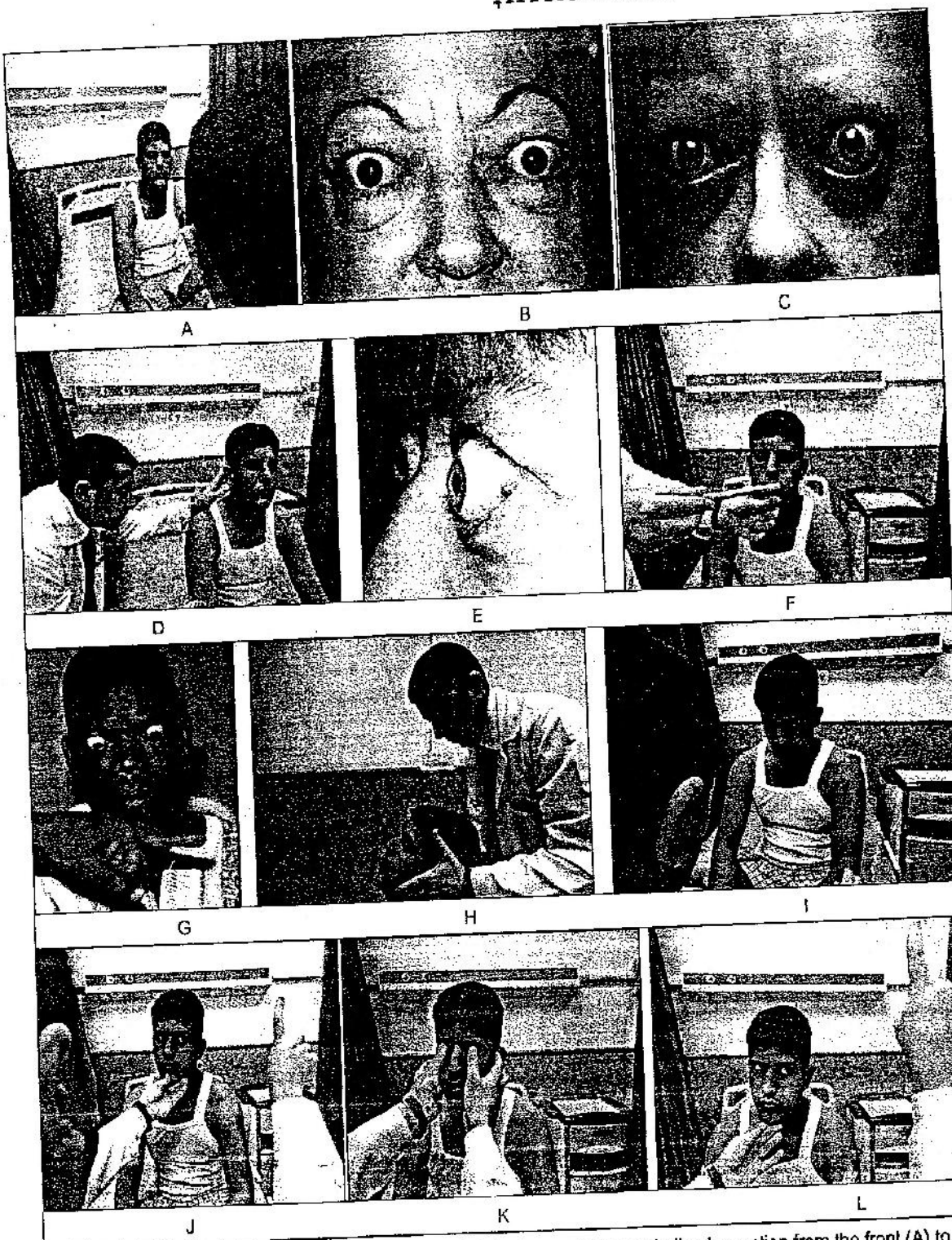


FIGURE 9 – 3. Examination of the eyes during thyroid gland examination including inspection from the front (A) to detect lid retraction (B) or exophthalmos (C); inspecting from the side (D) for exophthalmos (E) and to perform von Graefe's test (F) for lid lag (G); inspecting the eyes from behind above the patient head (Naffziger's method) (H); inspecting again from the front to perform (Joffroy's sign) (I), looking for chemosis and corneal ulceration (J), applying light pressure (K), and performing H test (L) as well as near accommodation test.

LOCAL EXAMINATION OF THE THYROID GLAND

The following steps should be considered (FIGURE 9 – 4):

INSPECTION

1. Stand in front of the patient to see his/her neck to detect any lump (swelling). In obese and short necked female, use the pizzillo's method. In which, the patient is asked to throw her head backwards and pressing her occiput against her clasped hands
2. Inspect neck veins if distended which may indicate a mass obstructing the thoracic inlet
3. Ask the patient then to swallow and if she can not, give her some water. The swelling will rise on swallowing because of the attachment of the thyroid to the larynx, but if the swelling persists fixed, this may indicate neoplasm or an inflammation
4. Then ask the patient to protrude his/her tongue so as to differentiate thyroid swelling from thyroglossal cyst where the latter will appear on protrusion of the tongue

PALPATION

I Palpation of the neck from the front

1. Palpate the gland from the front of the patient
2. Position of the trachea should also be ascertained
3. Palpate the posterior group of the cervical lymph nodes
4. Detect the carotid pulsation. Normally it is behind the anterior border of the sternomastoid muscle at the level of upper border of thyroid cartilage. Displacement of the carotid pulsation laterally occurs when there is a mass in the thyroid gland but its fixation in its position despite thyroid mass may suggest a malignancy (Berry's sign)

II Palpation of the neck from behind

1. Stand behind the seated patient and place your thumbs on the ligamentum nuchae and tilt the head slightly forwards to relax the anterior neck muscle. Both hands are placed with the pulps of the fingers over the gland. Feel systematically both lobes of the gland and its isthmus. Normally, the thyroid gland is not palpable. If the thyroid gland is difficult to be palpated, press over one lobe to feel the opposite one. Consider the followings:

   Size: only an approximate estimation is possible. Feel particularly carefully for a lower border, because its absence suggests retrosternal extension. Indeed a patient with retrosternal goiter may have no obvious lump as this lump will be pulled into the thoracic inlet due to negative thoracic pressure especially in athlete male. If the thyroid is large enough, it may impair venous outflow from the head and neck and may even be responsible for airway or vascular compromise.

   Pemberton's sign is a useful maneuver for detecting latent obstruction in the thoracic inlet due to retrosternal mass. To determine whether the sign is present, the patient is asked to elevate both arms until they touch the sides of the head. If the test is positive, indicating that Pemberton's sign is present, facial congestion (plethora) and cyanosis with dilatation of the cervical veins will develop within a few seconds.

   Shape: note whether the gland is uniformly enlarged or irregular and whether the isthmus is affected. If a nodule that feels distinct from the remaining thyroid tissue is palpable, determine its location, size, consistency, tenderness and mobility. Also decide whether the whole gland feels nodular (multinodular goiter)

   Consistency: may vary in different parts of the gland. Soft is normal; the gland is often firm in simple goiter and typically rubbery hard in Hashimoto's thyroiditis. A stony, hard node suggests carcinoma, calcification in a cyst, fibrosis or Riedel's thyroiditis

   Tenderness: a feature of thyroiditis or less often of a bleed into a cyst or carcinoma

   Mobility: carcinoma may tether the gland

2. Ask the patient then to swallow to see whether the lump is moving or fixed.
3. Palpate the whole cervical and supraclavicular lymph nodes

PERCUSSION

Percuss over the lower parts of the gland, along the clavicles, and over sternum and upper chest wall. Dullness may indicate retrosternal goiter.

AUSCULTATION

Listen over each lobe for a bruit. This is a sign of increased blood supply which may occur in hyperthyroidism, or occasionally from the use of antithyroid drugs. The differential diagnosis also includes a carotid bruit (louder over the carotid itself) or a venous hum (obliterated by gentle pressure over the base of the neck).

FIGURE 9 – 4. Examination of the thyroid gland including inspection from the front (A), using pizzillo's method (B), and with the protrusion of the tongue (C); palpation from front (D) and from behind the patient (E); performing Pemberton's sign (F and G); percussion (H) and auscultation (I).

Specific features of grave's disease are shown in BOX 9 – 2.

BOX 9 – 2. Features of Grave's disease

Goiter with bruit	Chemosis
Pretibial myxoedema	Exophthalmos
Finger clubbing (thyroid acropachy)	Periorbital oedema
Vitiligo	Corneal ulceration
Diplopia	Ophthalmoplegia
Loss of visual acuity	Papilloedema

If the order of examination was to examine the thyroid status of the patient, consider the following in addition to the above mentioned steps:

1. Deep tendon reflexes (hyper-reflexia with clonus in hyperthyroidism and delayed relaxation phase in hypothyroidism)
2. Gait (cerebellar ataxia in hypothyroidism)
3. Ask the patient to stand from seated position (proximal myopathy in hyperthyroidism and hypothyroidism)

EXAMPLES

Examine the thyroid status of this patient

Young aged conscious female patient sitting comfortably in her bed; she was not nervous, agitated, irritable, or dyspneic with normal body built. She had normal (cold or hot and this depends on the weather) hand temperature with no palmar erythema, and no nail changes; her radial pulse is 75 bpm regular rhythm with good volume, and no collapsing pulse; she had bilateral fine tremor. No hair loss had been noticed; she had no lid retraction, no exophthalmos, no lid lag, and no proptosis. She had impalpable thyroid gland with no lymphadenopathy, and centrally located trachea; no thyroid bruits is heard. She had normal leg texture; no myxoedema or edema; she had normal deep tendon reflexes, normal gait, and no proximal myopathy.

What is the interpretation?

The interpretation is normal thyroid status examination

Young aged conscious female patient sitting irritably in her bed; she was nervous and agitated and having thin body built. She had warm sweaty hands with palmar erythema, and onycholysis; her radial pulse is 110 bpm, irregular rhythm with large volume, and collapsing pulse; she had bilateral coarse tremor. No hair loss had been noticed; she had bilateral exophthalmos and lid lag. She had palpable thyroid gland with no lymphadenopathy; the thyroid gland was symmetrically enlarged non-tender, mobile and soft in consistency. The trachea was centrally located; negative Pemberton's sign; thyroid bruits is heard over both thyroid lobes. She had normal leg texture; no pretibial myxoedema or edema; she had hyer-reflexia (+++) of her deep tendon reflexes. She had proximal myopathy with no cerebellar ataxia.

What is the interpretation?

The interpretation is hyperthyroidism.

Young aged conscious female patient sitting in her bed; she was lethargic and obese. She had dry cold hands with no palmar erythema; her radial pulse is 50 bpm, regular rhythm with good volume. She had bilateral fine tremor. Her hairs were rough; she had no exophthalmos, no proptosis, and no lid lag. She had palpable thyroid gland with no lymphadenopathy; the thyroid gland was symmetrically enlarged non-tender, mobile and soft in consistency. The trachea was centrally located; negative Pemberton's sign; there was no thyroid bruits. She had cold rough legs with myxoedema; she had delayed relaxation phase of her ankle tendon reflexes. She had proximal myopathy with no cerebellar ataxia.

What is the interpretation?

The interpretation is hypothyroidism.

ANATOMY OF THE BREAST

The breasts are modified sweat glands that lie against the anterior thoracic wall, extending from the clavicle and second rib down to the sixth rib, and from the sternum across to the mid-axillary line. Its surface area is generally rectangular rather than round. Pigmented skin covers the areola and the nipple, which is erectile tissue. The openings of the lactiferous ducts are seen near the apex of the nipple. The nipple is in the 4th intercostal space in the midclavicular line, but accessory breast/nipple tissue may develop anywhere down the nipple line (axilla to groin).

FIGURE 10 – 1. Accessory nipple.

Accessory breasts may exist as glandular tissue, nipple, or only the areola. The axilla is the most common site for these anomalous structures, followed by a site just below the normal breast (FIGURE 10 – 1). In more than 50% of all patients with accessory breast tissue, the anomalies are bilateral. In general, accessory breast tissue is of little clinical significance. It usually has no physiologic function and is rarely associated with disease. The adult breast (FIGURE 10 – 2) is divided into the nipple, the areola and four quadrants, upper and lower, inner and outer, with an axillary tail projecting from the upper outer quadrant.

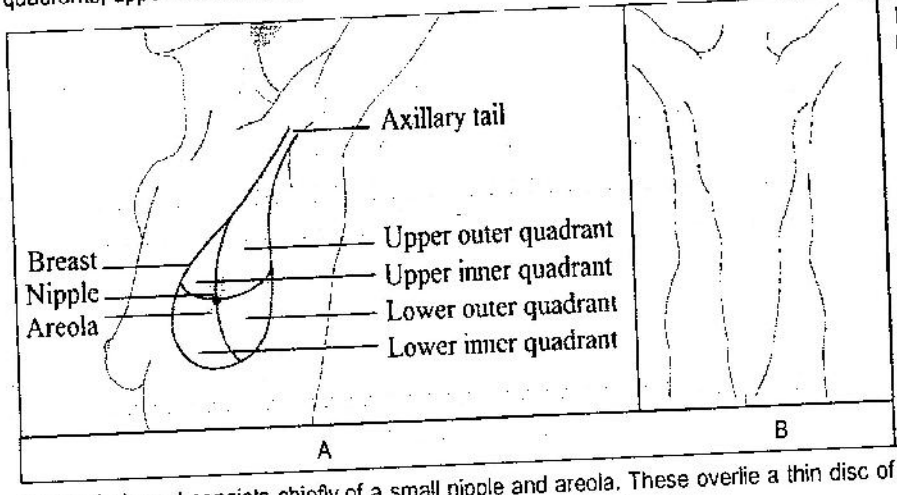


FIGURE 10 – 2. Divisions of adult female breast (A) and the nipple line (B).

The male breast consists chiefly of a small nipple and areola. These overlie a thin disc of undeveloped breast tissue that may not be distinguishable clinically from the surrounding tissues.

EXAMINATION OF THE BREAST

Some considerations should be followed:

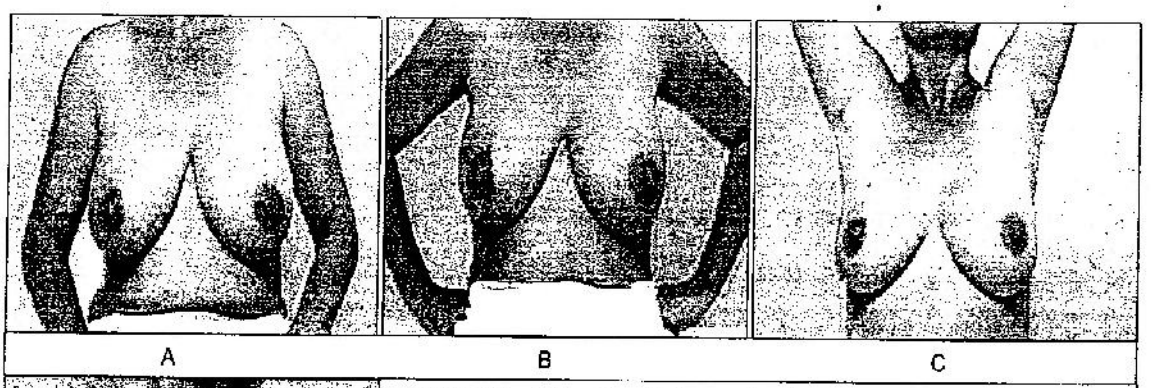
1 The patient should be undressed completely to the waist
2 Initially, the patient should seat on a chair. The candidate should sit opposite to the patient and if male, patient's relative should be present
3 Always, axillary lymph nodes should be examined as a part of breast examination

INSPECTION

Ask the patient to undress to the waist and sit upright on a well-illuminated chair and:

1 Ask her to rest her hands on the thighs to relax the pectoral muscles (POSITION I: FIGURE 10 – 3 A). Face the patient and look at the breasts for the seven Ss and seven Ds clinical findings described below.
2 Ask the patient to press her hands firmly on the hips to contract the pectoral muscles and repeat the inspection (POSITION II: FIGURE 10 – 3 B).
3 Ask her to raise her arms above the head and then to lean forward to expose the whole breast and exacerbate skin dimpling (POSITION III AND IV: FIGURE 10 – 3 C and D).

Dimpling or retraction of the breasts/nipples/areola in the last three positions suggests an underlying cancer. The last position may reveal an asymmetry of the breast or nipple that is not visible with other positions.



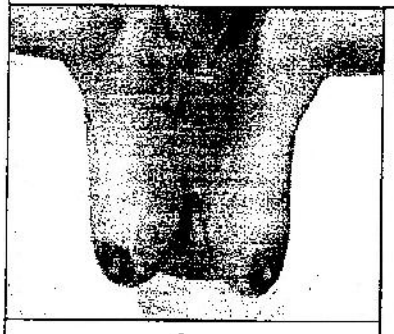


FIGURE 10 – 3. Positions for inspecting the breasts. (A) Hands resting on thighs. (B) Hands pressed onto hips. (C) Arms above head. (D) Leaning forward with breasts pendulous.

To describe clinical findings, the breast is often divided into four quadrants based on horizontal and vertical lines crossing the nipple (FIGURE 10 – 4). An axillary tail of breast tissue extends toward the anterior axillary fold. Alternatively, findings can be localized as the time on the face of a clock for example 3 o'clock and the distance in centimeters from the nipple.

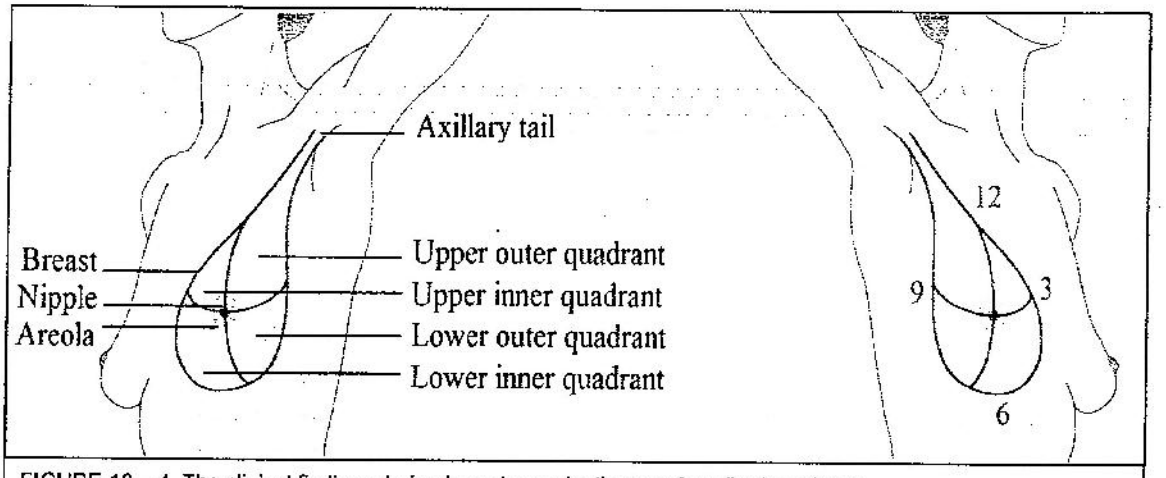


FIGURE 10 – 4. The clinical findings during breast examination are described as shown.

Carefully look for the seven Ss and the seven Ds of the breast (FIGURE 10 – 5). These are:

S1 Size of both breasts. Normally, the right breast is larger than the left

S2 Symmetry. Normally the left breast usually lies somewhat lower than the right

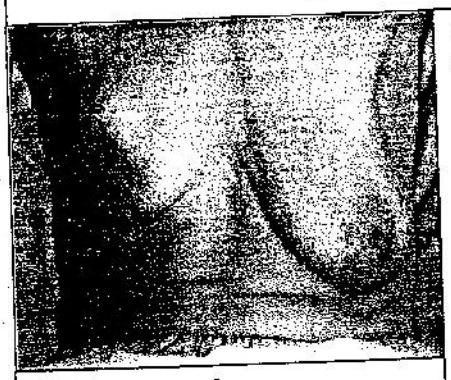
S3 Skin of the breast should be inspected for ulceration, discoloration, thickening, and nodularity. Red color may suggest eczema or infection. Discoloration, thickening, or nodularity may indicate infiltration with malignancy. Peau d'orange is the term used to describe alteration in the quality of the skin where the skin is attached at the hair follicles, but swollen in between, giving the appearance of the skin of an orange (FIGURE 10 – 5)

S4 Superficial visible veins coursing over both breasts are not diagnostic, but such veins if present only over one breast it means that there is active lesion in that breast

S5 Swelling in the areola indicates retention cyst of Montgomery

S6 Seven Ds of the nipple:

A B



C

FIGURE 10 – 5. Skin dimpling due to underlying malignancy (A); peau d'orange breast skin (B); asymmetrical breasts (C).

D1 Destruction

D2 Depression (retention or inversion). The characteristics of benign nipple inversion is a symmetrical slit-like inversion, but nipple retraction due to malignant disease is a symmetrical and distorting, pulling the nipple away from it's central position

D3 Discoloration

D4 Displacement which indicates ectopic nipple

D5 Deviation

D6 Duplication

D7 Discharge. Red (bloody) discharge occurs in duct papilloma, duct carcinoma and ductectasia; yellow (serous or pus) discharge occurs in duct papilloma or abscesses; green (serous and debris) discharge indicates ductectasia; white discharge indicates milk

S7 See the axillae for any swelling, ulceration or puckering after asking the patient to slowly raise her hands over her head

PALPATION

I. PALPATION OF THE BREASTS

The patient should lie in supine position because palpation is best performed when the breast tissue is flattened. Ask the patient to point the abnormal breast in order to palpate the normal one because the texture of the breast varies from female to another. The palpation is performed by the finger pads of the second, third, fourth fingers, keeping the fingers slightly flexed. The palpation should be performed in systematic (FIGURE 10 – 6) manner. Compress the breast tissue firmly against the chest wall and compare the texture of one breast with the other. Examine the axillary tail between your fingers and thumb as it extends towards the axilla. The following points need to be carefully elucidated if a lump is detected (mnemonic: SPACES - I):

1	Size and shape	Approximate size and shape
2	Position	According to FIGURE 10 – 4
3	Attachments	To the skin (tethering or fixation) or to the muscles

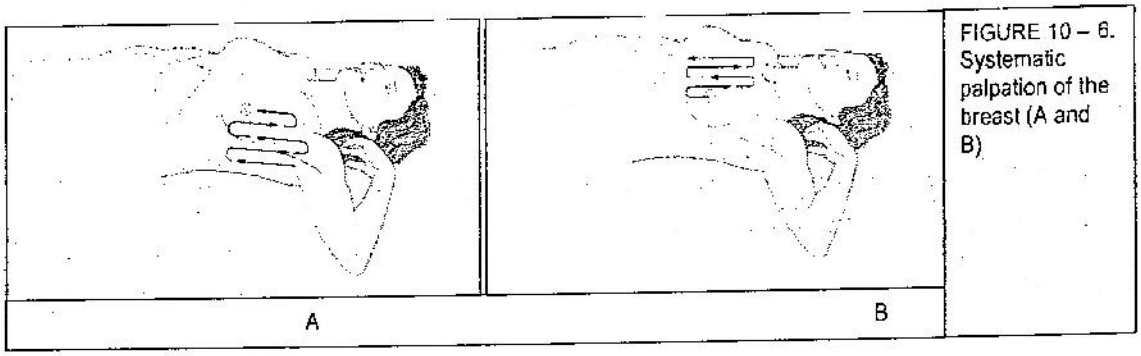


FIGURE 10 – 6. Systematic palpation of the breast (A and B)

ATTACHMENT TO THE SKIN

A lump may be described as tethered to the skin if it can be moved independently of the skin for a limited distance but pulls on the skin if moved further. To demonstrate tethering, move the lump from side to side and look for skin dimpling at the extremes of movement. In skin fixation, the lump and the skin overlying it cannot be moved independently.

ATTACHMENT TO THE MUSCLES

With the patient's hands on her hips, hold the mass between your thumb and forefinger. Ask her to contract and relax the pectoral muscles alternately by pushing into her hips. As the pectoral muscle contracts, note whether the mass moves with it and if it is separate when the muscle is relaxed. Infiltration suggests malignancy.

A swelling towards the periphery of the lower outer quadrant lie on the serratus anterior thus ask the patient to place the hand of the affected side upon your shoulder and to press. This will result in contraction of the serratus anterior muscle. Now re-estimate the mobility as above.

4	Consistency	Hard, irregular nodule is characteristic of carcinoma
5	Edge	The edge or margin may be well delineated or ill defined
6	Single or multiple	Multiple nodules suggest benign cystic disease or fibroadenosis
7	Inflammation	Redness, tenderness, and warmth

II. PALPATION OF THE NIPPLES

Palpate the nipple by holding it gently between your index finger and thumb. Try to express any discharge. Massage the breast towards the nipple to uncover any discharge.

III. PAPATION OF THE CERVICAL AND AXILLARY LYMPH NODES

The techniques for lymph nodes palpation are described under the general examination heading.

EXAMINATION OF THE ARMS

Examine the arm for any evidence of swelling, arterial and venous abnormalities, and neurological abnormalities.

Presentations of breast disease are shown in BOX 10 – 1.

BOX 10 – 1. Presentations of breast disease

Painless lump	Pain and tenderness but not a lump	Change in the breast size
Carcinoma Fat necrosis Cystic hyperplasia Fibroadenoma	Premenstrual tension Mild cystic hyperplasia Pregnancy mastitis	Carcinoma Pregnancy Benign hypertrophy Giant fibroadenoma
Painful lump	**Changes in the nipple**	**Discharge**
Carcinoma Fat necrosis Cystic hyperplasia Abscess	Carcinoma Duct papilloma Paget's disease of the nipple Eczema	Duct carcinoma Duct papilloma Abscesses Ductectasia

SURGICAL ANATOMY OF HERNIA

Hernia is a protrusion of a viscus or part of a viscus through an abnormal opening in the walls of its containing cavity. Causes of hernia are provided in BOX 11 – 1.

BOX 11 – 1. Causes of hernia

Congenital

1 Congenital preformed sac as in indirect inguinal hernia. In male, the cause is persistent processus vaginalis while in female this is because of patency of canal of nuck
2 Failure of umbilical scar to be formed as in true umbilical hernia
3 Persistent communication between the abdominal cavity and the thoracic cavity as in hiatus hernia

Acquired

1 Straining on micturition e.g. benign prostatic hyperplasia, urethral stricture, and bladder neck obstruction
2 Straining on defecation for example chronic constipation
3 Chronic cough e.g., asthmatic patient and chronic bronchitis
4 Stretching of abdominal musculature as in obesity
5 Pregnancy and ascites
6 Wasting of abdominal muscles as in cachexia, normal aging process and paresis as in poliomyelitis

Iatrogenic as in incisional hernia

As shown in FIGURE 11 – 1, the hernia consists of:

1 The sac

The sac is a peritoneal diverticulum and divided into mouth, body, fundus and neck

2 The coverings of the sac

These are derived from the layers of the abdominal wall through which the sac passes

3 The contents of the sac

Common types of herniae are shown in BOX 11 – 2.

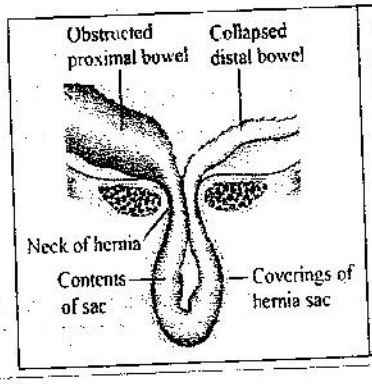


FIGURE 11 – 1. Anatomy of hernia.

BOX 11 – 2. Types of hernia

Common herniae

1 Epigastric hernia
2 Paraumbilical hernia
3 Umbilical hernia
4 Incisional hernia
5 Femoral hernia
6 Inguinal hernia which is of two types:
   A Direct inguinal hernia
   B Indirect inguinal hernia

Other rare herniae

1 Spigelian hernia
2 Obturator hernia
3 Gluteal hernia
4 Lumber hernia

Hernia is surgically classified into:

1 Reducible hernia	The contents of such a hernia can be reduced by the patient. This hernia has also an expensile cough impulse
2 Irreducible hernia	The contents of such a hernia cannot be reduced by the patient due to adhesion between the contents and the sac with no evidence of other complication
3 Obstructive	It is irreducible hernia, but due to obstruction from inside to outside with no interference to the blood supply
4 Strangulated hernia	When there is interference with the blood supply, the irreducible hernia is termed strangulated
5 Inflamed hernia	This type of hernia occurs due to inflammation of the contents of the sac for example acute appendicitis

ANATOMY OF THE GROIN (FIGURE 11 – 2)

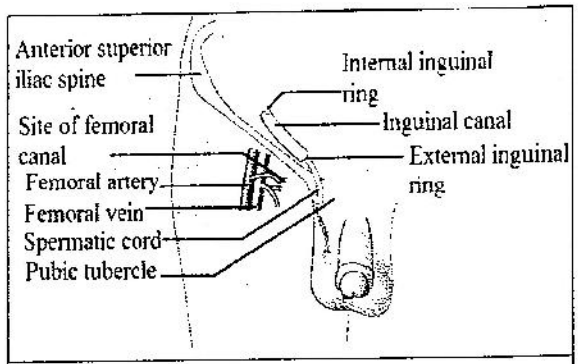


FIGURE 11 – 2. Anatomy of the groin region.

Because hernias are relatively common, it is important to understand the anatomy of the groin. The basic landmarks are the anterior superior iliac spine, the pubic tubercle, and the inguinal ligament that runs between them. The inguinal canal (lies above and approximately parallel to the inguinal ligament) forms a tunnel for the vas deferens as it passes through the abdominal muscles. The exterior opening of the tunnel, the external inguinal ring, is a triangular slit like structure palpable just above and lateral to the pubic tubercle. The internal opening of the canal, internal inguinal ring, is about 1 cm above the midpoint of the inguinal ligament. Neither canal nor internal ring is palpable through the abdominal wall. When loops of bowel force their way through weak areas of the inguinal canal they produce inguinal hernias. Contents of the inguinal canal in male are the spermatic cord and the ilioinguinal nerve while in female the contents are round ligament and ilioinguinal nerve. The spermatic cord contains 3 coverings, 3 vessels, and 3 other structures. The coverings are the external spermatic fascia, the cremastric fascia and muscle, and the internal spermatic fascia. The vessels are vas deference, testicular artery, and testicular vein. Other structures are the lymph vessels, autonomic nerve, and processus vaginalis. The sac of the indirect inguinal hernia begins at the internal ring and is inside the spermatic cord. The sac of direct inguinal hernia appears out side spermatic cord.

Another potential route for a herniating mass is the femoral canal. This lies below the inguinal ligament. Although you can not see it, you can estimate its location by placing your right index finger, from below, on the right femoral artery. Your middle finger will then overlie the femoral vein; your ring finger, the femoral canal. Femoral hernias protrude here.

SURGICAL ANATOMY OF GROIN HERNIA

Groin hernia consists of inguinal hernia (direct and indirect) and femoral hernia. The internal (deep) inguinal ring is a fibrous ring of transversalis fascia, u– shape, and is about 1 cm above mid-inguinal point. The external (superficial) inguinal ring s a condensation of the aponeurosis of the external oblique muscle, inverted V–shape, and is about 1cm above the pubic tubercle. Between these two rings is the inguinal canal which is oblique canal except in infants where the internal and external rings are superimposed. Mid-inguinal point is equidistance between anterior superior iliac spine and the symphysis pubis. The inguinal canal is bounded anteriorly by the skin, superficial fascia, external oblique and internal oblique aponeurosis (roof); posteriorly (floor) is bounded by the conjoined tendon (represent the common fused insertion of the internal oblique and transversalis abdominis muscles into the pubic crest) on the medial side and on the lateral side it is bounded by inferior epigastric artery; superiorly (above) the canal is bounded by conjoined tendon and inferiorly (below) by inguinal ligament.

Femoral hernia is commonly present in females because femoral canal is wider than that in males. Because of the tight ligament in the femoral canal, femoral hernia is more liable to be obstructive or strangulated. The femoral ring is about 1 inch below and lateral to pubic tubercle. The femoral canal is bounded anteriorly by the inguinal ligament; posteriorly is bounded by the pectineal ligament (ligament of Cooper); medially is bounded by the sharp edge of the pectineal part of the inguinal ligament and laterally by the the femoral vein.

EXAMINATION OF GROIN HERNIA

Examination sequence of groin hernias should include:

INSPECTION

Ask the patient to stand up and inspect any swelling. This step should include both inguinal regions. Ask the patient then to turn his head laterally and cough (to avoid salivary shower). Normally in thin individual, there is a small bulging in both inguinal regions (Malgaignes bulgings), but those with hernia, the bulging is usually big enough to be noticed. Upon inspection decide the following:

1. The bulging starts to appear from above inguinal ligament laterally and descend into the scrotum so it is more likely indirect inguinal hernia
2. The bulging appears above inguinal ligament medially and did not descend into the scrotum so it is more likely direct inguinal hernia
3. The bulging appears below the inguinal ligament and below and lateral to the pubic tubercle and the patient is female so it is more likely femoral hernia
4. The bulging appear from the site of a previous scar e.g., appendicectomy scar so it is incisional hernia

SCROTAL PALPATION

If there is obvious swelling in the scrotum, grasp the swelling (FIGURE 11 – 3) between your thumb and the index finger and decide whether it is possible to get above the swelling so it is not hernia, but if you cannot get above it so it is more likely to be a hernia.

INVAGINATION TEST

This test is used to differentiate the inguinal from femoral hernia. The index finger (FIGURE 11 – 3) is introduced into the inguinal canal; if the lump is present or it appears when the patient is asked to cough so it is inguinal hernia. If the hernia examination is performed with adequate scrotal skin and is done slowly, it is painless.

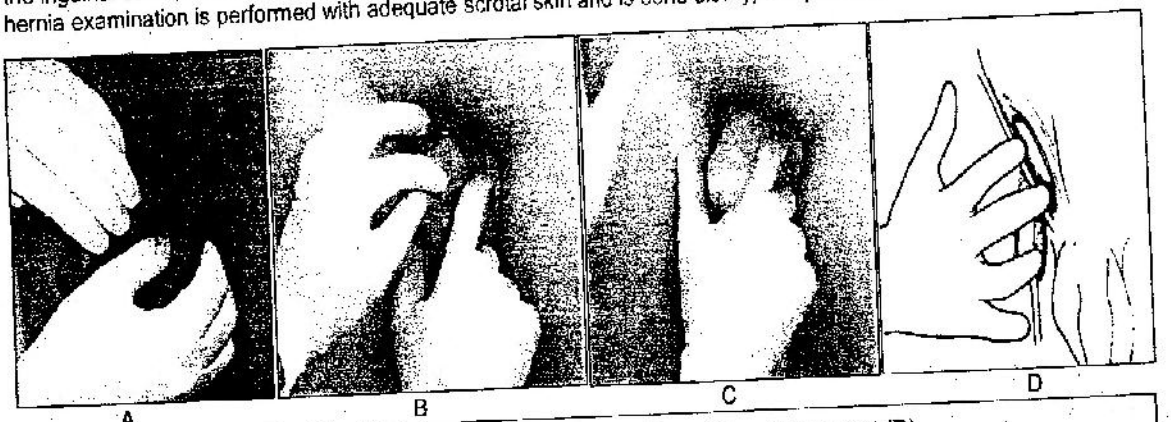


FIGURE 11 – 3. Scrotal palpation (A), invagination test (B and C), and three fingers test (D).

LUMP PALPATION

Place your hand flat on the groin parallel to the inguinal ligament and your other hand on the patient's back to stop him pushed over. The lump is then analyzed as follows:

Size	May by small or large. Its length, width and depth are measured by tape measure
Position	At the right or left iliac fossa or at the femoral ring
Attachment	Usually not attach to skin or muscle unless complications occur
Consistency	Usually soft or firm
Edge	Well defined edge
Surface and shape	Smooth regular surface and pear or sphere in shape with long stalk
Pulsation, thrills, and bruits	Negative
Inflammatory signs	May be present if inflamed
Transillumination	Usually negative
Multiplicity	May be unilateral or bilateral
Composition	The contents of inguinal hernia are loops of bowel
Compressibility	Negative since the hernia the lump will not reappear without effect of cough or gravity
Reducibility	Ask the patient to reduce hernia by him self and then follow his steps of reducibility. A lump reduces to a point above and medial to pubic tubercle is inguinal while reduction to a point below and lateral to pubic tubercle suggests femoral hernia. Since there is reducibility, you must perform expansile cough impulse test by asking the patient to turn his head laterally and cough while your hand compressing the swelling. If the swelling expands with the cough, it has cough impulse which is almost diagnostic for hernia but if there is movement of the swelling without expansion or an increase in the tension, so it is not a cough impulse, however, absence of expansile cough impulse does not exclude hernia because hernia may by obstructed or strangulated.
Resonance	May be negative when small or positive when very large
Fluctuation	Usually negative

OCCLUSION TEST

Also called control test. When you reduce the hernia through the external ring slide your hand laterally keeping it at the internal ring then ask the patient to cough; if no hernia reappears so it is indirect otherwise it is direct.

ZIEMAN'S TEST (THREE FINGERS TEST)

Place the index finger over the indirect (deep ring), middle finger over the direct (superficial ring) and the ring finger over the femoral ring (FIGURE 11 – 3). The patient now is asked to close his nose and blow; there will be a motion or pressure sensation at one of these three sites if there is hernia. More over, by removal of one finger and asking him to blow, there will be appearance of a lump; if not, remove another finger and so on.

REAPPEARANCE TEST

After reducing the hernia, wait and watch the way of reappearance that will decide the type of the hernia.

CLINICAL TERMS OF HERNIAS
Hernias can be termed according to their contents into:

1	Richter's hernia	Part of the circumference of the intestine in the hernial sac
2	Litter's hernia	Hernial sac contains Meckel's diverticulum
3	Maydl's hernia	Two loops of the bowel in the hernial sac
4	Sliding hernia	When an organ or part of it which slides down into the hernial sac. On the right side, the most likely organ is the cecum and on the left side is the urinary bladder
5	Omentocele	Hernial sac contains an omentum
6	Enterocele	Hernial sac contains small intestine

The differential diagnoses of groin lumps are provided in BOX 11 – 3.

BOX 11 – 3. Differential diagnoses of groin masses

Inguinal hernia	Ectopic testis
Femoral hernia	Femoral hernia
Vaginal Hydrocele	Inguinal hernia
Encysted hydrocele of the cord or canal of nuck	Saphena varix
Undescended testis	Enlarged lymph nodes
Lipoma of the cord	Lipoma
Spermatocele	Psoas abscess
Scrotal swelling e.g., tumour and hematocele	Psoas bursa
Ruptured adductor longus with hematoma	Femoral artery aneurysm
Hydrocele of femoral hernial sac	

Complications of inguinal hernia include adhesion, obstruction, strangulation, and inflammation. Clinical comparison of both types of inguinal hernia is provided in BOX 11 – 4.

BOX 11 – 4. The clinical features of the inguinal hernia

Indirect hernia	Direct hernia
Can descend into the scrotum	Cannot descend into the scrotum
Reduces upwards then laterally and back wards	Reduces upwards then straight back wards
Controlled by pressure over internal ring	Controlled by pressure over external ring
The defect is not palpable because it is behind fibers of external oblique muscle	The defect may be felt in the abdominal wall, above the pubic tubercle
After reduction, the bulge reappears in the middle of the inguinal region then flows medially before turning down to the neck of the scrotum	After reduction, the bulge reappears exactly where it was before
Protrusion through deep inguinal ring	Protrusion through posterior wall of inguinal canal
Congenital and acquired and occurs frequently in children and not rare in females	Usually acquired in men over 40 years. It is never occur in children and rare in females
85%	15%
Lateral to inferior epigastric artery	Medial to inferior epigastric artery
Usually unilateral and strangulation is a common complication	Usually bilateral and rarely strangulated due to wide neck

LUMP/SWELLING EXAMINATION

The clinical examination of a lump often provides a narrow range of differential diagnoses or even a definite diagnosis. A useful mnemonic for the elements of a lump examination is SPACE SPIT MCRF:

S SIZE

Avoid use misleading terms e.g. large, medium or small instead use tape measure to measure the length, width and depth if possible.

P Position

The position of the swelling should be described in its exact anatomical location.

A ATTACHMENTS

Lymphatic obstruction causes fixation of the skin with fine dimpling at the opening of hair follicles that resembles orange peel (peau d'orange). This is common in malignant disease. Fixation to deeper structures may occur in a breast cancer fixed to underlying muscle. A lump attached to the skin should be tested gently and over the whole lump. When a lump attached to deep structure, it can be tested by moving the lump in two directions (horizontal and vertical). If the lump attaches to the muscle, let the muscle contracts and again move the lump in two directions. If the lump moves, so it is not attached to the muscles. To detect a lump in the abdomen whether intra-abdominal e.g., in the abdominal cavity or in the abdominal wall, ask the patient to lift his head and shoulders off the pillows then press firmly against his forehead to contract abdominal wall muscle. If the swelling disappears, it is within abdominal cavity. If the swelling becomes more prominent, it is within abdominal wall.

C CONSISTENCY

The whole lump should be examined and described as very soft (like Jelly), soft (as relaxed muscle), firm (as contracted muscle), hard (as contracted biceps of a boxer), stony hard (like a stone), rubbery (like a rubber), or spongy (like a sponge).

E EDGE

The margin of a swelling may be well defined or ill defined.

S SURFACE AND SHAPE

The texture of the mass may vary from uniformly smooth and regular to grossly irregular or nodular. Use descriptive terms for shape e.g. pear-, kidney-, or sphere-shaped.

P PULSATION, THRILLS, AND BRUITS

Pulsation implies arterial aneurysms or highly vascular tumours. Other swellings may be pulsatile if they lie over a major blood vessel. You must determine whether the lump is a truly pulsatile mass or false pulsatile i.e. got the pulsation from near by pulsatile structure. There are two methods:

Current sign	By placing a finger of each hand on opposite sides of the lump and feel if they are being pushed a part so it is a true expansile pulsation or pushed in the same direction (usually upward) so it is a transmitted pulsation (FIGURE 12 – 1)
Knee – elbow position	If the lump is found in the abdomen, ask the patient to adopt a knee – elbow position and see the lump; disappearance of pulsation suggests transmitted (false) pulsation

If the blood flow through a lump is increased, a systolic murmur (bruit) may be heard and, if loud enough, a thrill may be palpable. Bruits are also heard over arterial aneurysms and arteriovenous malformations.

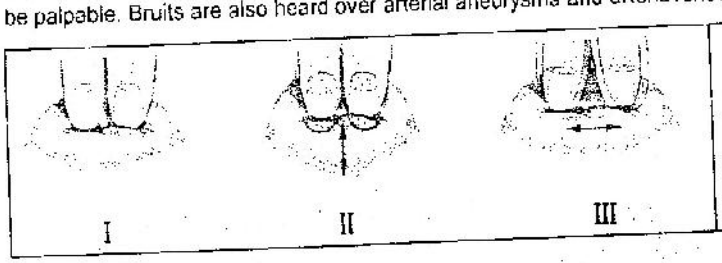


FIGURE 12 – 1. Detecting an expansile impulse where in I = no impulse; in II = transmitted pulsation from a neighboring artery; and in III = expansile impulse, the sign of an aneurysm.

I INFLAMMATION SIGNS

Redness, tenderness, and warmth suggest underling inflammatory lump.

T TRANSILLUMINATION

Transillumination is tested by light torch in a quite dark room. Presence of transillumination suggests fluid like water, lymph, plasma or serous fluid. No transillumination suggests solid, blood or other opaque fluid.

M MULTIPLICITY

Determine whether there are multiple lumps or single lump.

C COMPOSITION

Any lump should have some of the following features; solid, fluid, or gas.

C COMPRESSIBILITY

Some lumps when compressed, they will disappear and when the hand removed, they reappear again. Common causes are hemangioma, capillary angioma, and meningocele.

R REDUCIBILITY

Reducibility is a feature of hernia so you have to perform expansile cough impulse for a lump having reducibility. You must differentiate between reducibility and compressibility where in compressibility, the lump will re-appear from the same site under no effect for cough or gravity while reducibility means the lump will reappear from a different site under effect of cough or gravity.

R RESONANCE

It is only possible to test on large lumps. Percuss as you would any other part of the body and listen (and feel) if the lump is hollow (gas-filled) or solid.

F FLUCTUATION

Press one side of the lump the other sides may protrude. If the lump is solid, it will bulge at the opposite side only.

SLIPPING SIGN
If the edge of the lump is pressed, the swelling slips from beneath the finger. It is pathognomonic of a subcutaneous lipoma (FIGURE 12 – 2).

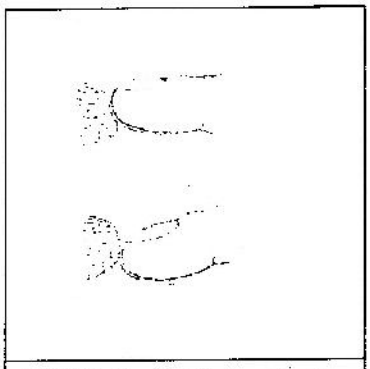


FIGURE 12 – 2. Slipping sign to detect lipoma.

ULCER EXAMINATION

Ulcer means discontinuation of the epithelial tissue due to loss of the whole epidermis and at least the upper part of the dermis healed by scar tissue. Main causes of leg ulcers are provided in BOX 13 – 1. The three main types of ulcer are venous, arterial, and neuropathic ulcers.

BOX 13 – 1. Causes of leg ulceration

Venous hypertension

There is a superficial low pressure venous system connected to a deep high pressure veins by perforating veins resulting in retrograde flow of the blood to the superficial system (venous hypertension) and consequently ulceration

Arterial diseases	Abnormalities of blood		Neuropathy
Atherosclerosis	Sickle cell disease		Diabetes mellitus
Vasculitis	Cryoglobulinaemia		Syphilis
Buerger's disease	Spherocytosis		Leprosy
Small vessels diseases	Tumour		Trauma
Diabetes mellitus	Squamous cell carcinoma	Malignant melanoma	
Vasculitis	Basal cell carcinoma	Kaposi's sarcoma	

The clinical examination of a lump often provides a narrow range of differential diagnoses or even a definite diagnosis. A useful mnemonic for the elements of a lump examination is SPADE BITS:

S SIZE

The size of the ulcer should be recorded in three dimensional pattern; the length, width, and the depth.

P Position

The position of the ulcer should be described in its exact anatomical location.

A ATTACHMENTS

The ulcer is usually adhere to deep structures

D DEPTH

Depth should be measured in millimeters.

D DISCHARGE

Any discharge e.g. serous fluid, pus, or blood should be noted.

E EDGE

Various types of ulcer's edge are commonly present during clinical sessions. Generally speaking, the edge may fall into one of five types (FIGURE 13 – 1).

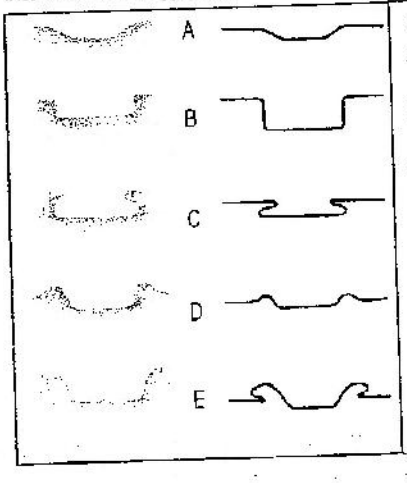


FIGURE 13 – 1. Types of ulcer edges.
A) Flat sloping ulcer edge. These ulcers are usually shallow and implies that it is healing (e.g. venous ulcers).
B) Punched out ulcer edge. This is full-thickness skin loss with no healing and typical of neuropathic ulceration and vasculitic lesions.
C) Undermined ulcer edge. These extend below the visible edge creating a lip. This is typical of pyoderma gangrenosum and infected ulceration such as tuberculosis.
D) Rolled ulcer edge. The edge is mounded but neither everted or undermined and implies proliferation of the tissues at the edge of the ulcer. This is typical for basal cell carcinoma.
E) Everted edge ulcer. Here the tissues at the edge of the ulcer are proliferating too fast, creating an everted lip. This is typical of neoplastic ulceration.

B BASE

If the ulcer's base can be seen (i.e. not covered with mucus, blood or crust), it should be carefully described. Ulcer usually has either slough or granulation tissue at the base. Look especially for bone, tendons, and blood vessels.

I INFLAMMATION SIGNS

Redness, tenderness, and warmth suggest inflammation.

T TEMPERATURE

Venous ulcer is usually warm while arterial ulcer is cold.

S SHAPE

The texture of the ulcer is described e.g. regular, irregular, or circular.
BOX 13 – 2 shows the clinical features of the main types of leg ulcers (FIGURE 13 – 2).

BOX 13 – 2. Clinical features of leg ulcers

Characteristics	Arterial (ischemic) ulcer	Venous ulcer	Neuropathic ulcer
Size	Any size	Any size	Any size
Position	It is found in the pressure areas e.g. malleoli, heel, fifth metatarsal base, metatarsal heads and toes	It is found in the inner and some times the outer ankle	It is found in pressure points in areas with diminished sensation e.g. heel, ball of the foot or toes
Attachment	The ulcer base may be attached to or may be a part of the underlying structures	The ulcer base may be attached to or may be a part of the underlying structures	The ulcer base may be attached to or may be a part of the underlying structures
Depth	Deep	Superficial	Deep and penetrating ulcer
Discharge	Serous exudates or purulent, but rarely sanguineous	Serous or purulent or sanguineous	Discharge is serous or purulent
Edge	Punched out ulcer edge	Flat sloping ulcer edge	Punched out ulcer edge
Base	Slough or granulation tissue	Slough or granulation tissue	Slough or granulation tissue
Inflammatory signs	Tenderness is the rule	Tenderness is present in third of cases	Painless
Temperature	Cold	Warm	Ambient temperature
Shape	Described accordingly	Described accordingly	Described accordingly

A B C

FIGURE 13 – 2. Main types of leg ulcers including arterial (A), venous (B), and neuropathic (C).

CHRONIC ULCER
The main causes of chronic ulcer are shown in BOX 13 – 3.

BOX 13 – 3. Causes of chronic ulcer

Infection and repeated trauma	Denervation
Ischemia and edema	Localized destructive diseases as in tuberculosis and carcinoma

This section focuses on circulation to the arms and legs. It includes the arteries, the veins, and the lymphatic system with its lymph nodes.

ARTERIES

Arterial pulses are palpable when an artery lies close to the body surface. In the arms, there are three arterial pulses (FIGURE 14 – 1). Pulsations of the brachial artery can be felt in and above the bend of the elbow, just medial to the biceps tendon and muscle. The brachial artery divides into the radial and ulnar arteries. Radial artery pulsations can be felt on the flexor surface of the wrist laterally. Medially, pulsations of the ulnar artery may be palpable, but overlying tissues frequently obscure them. The radial and ulnar arteries are interconnected by two vascular arches within the hand. Circulation to the hand and fingers is thereby doubly protected against possible arterial occlusion.

In the legs, arterial pulsations can usually be felt in four places. Those of the femoral artery are palpable below the inguinal ligament, midway between the anterior superior iliac spine and the symphysis pubis. The femoral artery travels downward deep within the thigh, passes medially behind the femur, and becomes the popliteal artery. Popliteal pulsations can be felt in the tissues behind the knee. Below the knee, the popliteal artery divides into two branches, both of which continue to the foot. There the anterior branch becomes the dorsalis pedis artery. Its pulsations are palpable on the dorsum of the foot just lateral to the extensor tendon of the big toe. The posterior branch, the posterior tibial artery, can be felt as it passes behind the medial malleolus of the ankle. Like the hand, the foot is protected by an interconnecting arch between its two chief arterial branches (FIGURE 14 – 1).

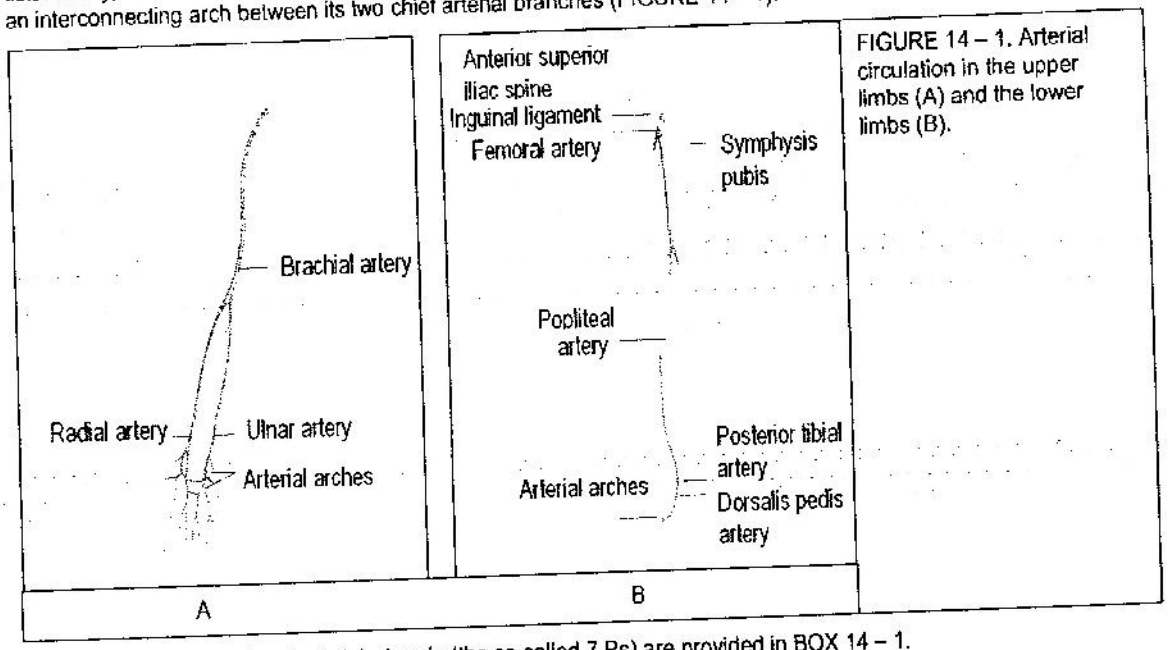


FIGURE 14 – 1. Arterial circulation in the upper limbs (A) and the lower limbs (B).

Symptoms and signs of acute limb ischemia (the so called 7 Ps) are provided in BOX 14 – 1.

BOX 14 – 1. Symptoms and signs of acute limb ischemia **7 Ps**

Symptoms/signs	Comment
Pain	May be absent in complete acute ischemia, and can be present in chronic ischemia
Pallor	
Pulselessness	
Perishing cold	Unreliable, as the ischemic limb takes on the ambient temperature
Paresthesia	Important features of impending irreversible ischemia
Paralysis	

EXAMINATION OF ARTERIAL CIRCULATION

The examination of arterial circulation includes:

INSPECTION

1 COLOR

In normal person, the color is usually pink in all positions, but in person with ischemic limb, the color may be pallor or even purple – blue cyanosed appearance

2 The vascular angle (Buerger's angle)

It is the angle to which the leg must be raised before it becomes white. Normally, the angle is 90° and the toes are pink, but in ischemic leg, the angle could be as low as 15°. Buerger's angle of < 20° indicates severe ischemia

3 Capillary filling time

It is the time taken for foot to become pink and depend on the degree of arterial obstruction. After estimation of the vascular angle, the patient is asked to sit up and hang legs down over the side of the couch. Normally the color on elevating up to 90° and after lowering feet is pink. In ischemic leg, the time will be as long as 15 – 30 seconds

4 Venous filling (Guttering of the veins)

The patient is asked to lie supine and raise the leg above the heart level. In normal person, the veins never collapse but in severe ischemic leg elevation of 10°–15° causes collapse of the veins

5 Pressure areas

Inspect areas which are subjected to pressure or trauma during walking or bed rest because they are the first sites that show trophic changes, thickening, blistering, purple or blue discoloration, ulceration and gangrene. These sites are the medial and lateral malleoli, skin of the head of the fifth metatarsal, toes tips, and webs of the toes

6 Inspect any deformities, pigmentation and swelling

PALPATION

1 Temperature

Assess the temperature by the back of the fingers as it is cool dry skin that is better than hot moist palmer surface

2 Capillary refilling

Press the tip of a nail or the pulp of a toe or finger for 2 seconds then observe the time taken for the blanched area to return pink after you have stopped pressing

3 Palpate the pulses

Feel the pulses

AUSCULTATION

1 Bruits

Listen over the bifurcation of the abdominal aorta below umbilicus, femoral artery, popliteal artery, posterior tibial and dorsalis pedis artery. Use the stethoscope bell pressed lightly

2 Blood pressure

Measure blood pressure of both arms to exclude subclavian stenosis

3 Reactive hyperemia

Inflate a sphygmomanometer cuff around the limb to 250 mmHg for 5 minutes then measure the interval between releasing the cuff and the appearance of red flush in the skin. In normal leg, it appears within 12 seconds but in severely affected limb, it may never appear. For this reason the candidate should take a permission

VEINS

The veins from the arms, together with those from the upper trunk and the head and neck, drain into the superior vena cava and on into the right atrium. Veins from the legs and the lower trunk drain upward into the inferior vena cava. Because the leg veins are especially susceptible to dysfunction, they warrant special attention. The deep veins of the legs carry about 90% of the venous return from the lower extremities. They are well supported by surrounding tissues. In contrast, the superficial veins are located subcutaneously, and are supported relatively poorly.
The superficial veins (FIGURE 14 – 2) include:

1 The great saphenous vein

The great saphenous vein originates on the dorsum of the foot, passes just in front of the medial malleolus, and then continues up the medial aspect of the leg to join the deep venous system (the femoral vein) below the inguinal ligament

2 The small saphenous vein

The small saphenous vein begins at the side of the foot and passes upward along the back of the leg to join the deep system in the popliteal space. Anastomotic veins connect the two saphenous veins superficially and, when dilated, are readily visible

In addition, communicating (or perforating) veins connect the saphenous system with the deep venous system.
Deep, superficial, and communicating veins all have one way valves. These allow venous blood to flow from the superficial to the deep system and toward the heart, but not in the opposite directions. Muscular activity contributes

importantly to venous blood flow. As calf muscles contract in walking, for example, blood is squeezed upward against gravity, and competent valves keep it from falling back again.

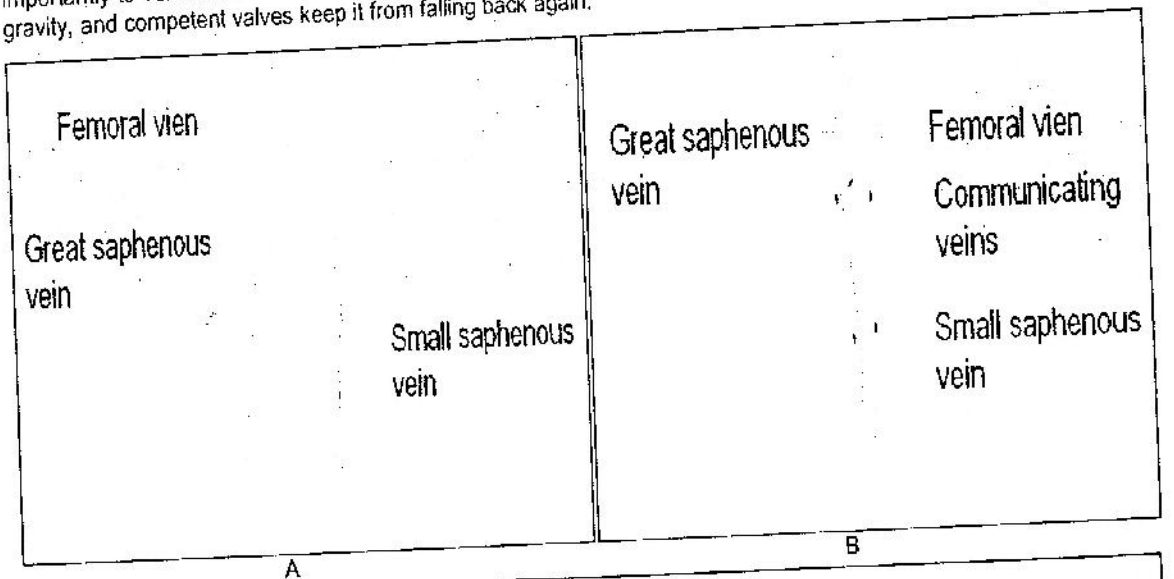


FIGURE 14 – 2. Lower limbs veins and the direction of blood flow in lower limb veins

EXAMINATION OF VARICOSE VEINS

Varicose veins (BOX 14 – 2) are tortuous, dilated veins that are usually having incompetent valves. Common veins involved are the long saphenous vein (LSV), the short saphenous vein (SSV), and their tributaries. Varicosity affects LSV seven times more than SSV. Both veins may be affected when their valves become incompetent or due to the generation of perforating veins. Any muscular activity therefore results in pushing of blood from deep to superficial instead from propelling the blood to the heart.

BOX 14 – 2. Causes of varicose veins

Primary

The cause is unknown but often familial due to weakness of the vein wall that permits valve ring dilatation or congenital absence of valve

Secondary

1. Due to obstruction to venous flow that occurs in:
   - Pregnancy, fibroid, and ovarian cyst
   - Abdominal lymphadenopathy
   - Pelvic cancer (cervix, uterus, ovary and rectum)
   - Ascites
   - Iliac vein thrombosis
   - Retroperitoneal fibrosis
2. Due to valve destruction as in deep venous thrombosis (DVT)
3. Due to high flow and pressure as in arteriovenous fistula

INSPECTION

With the patient standing, the lower limbs are inspected from the umbilicus to the toes. Remember to view the back of the leg as well as the front so as to avoid overlooking of varicosities of the short saphenous vein (FIGURE 14 – 3).

THE COUGH IMPULSE TEST

The fingers are laid on the thigh just below saphenous opening, in such a way that the pulps of the middle finger rests upon the vein. Then the patient is asked to cough. If the saphenofemoral junction is incompetent, a fluid thrill is felt by the finger. Thus a positive test indicates that the saphenofemoral valve (SFV) is incompetent.

THE BRODIE - TRENDELENBURG TEST

The patient lies down on a couch and the limb is raised to allow the blood to drain out the veins. The fingers or thumb are placed firmly over the saphenous opening. Keep firm pressing over saphenous opening while the patient standing. The hand is removed suddenly; if the veins fill immediately, the saphenofemoral valve is incompetent.

TOURNIQUET TEST

The patient lies down on a couch and the limb is raised to allow the blood to drain out the veins as the same as the previous test but now place a rubber tourniquet around the upper third of the thigh tight enough to occlude the superficial vein. Ask the patient now to stand up; if the veins above the tourniquet fill up but those below stay collapsed so there is incompetent valve along the vein above the tourniquet in addition to incompetent valve at saphenofemoral junction (SFJ).

CHEVRIER SIGN (PERCUSSION TAP)

Place the finger flat of one hand on the lower limit of visible veins and tap them at their upper limit. Palpable percussion impulses indicate dilated incompetent valves between these two sites.

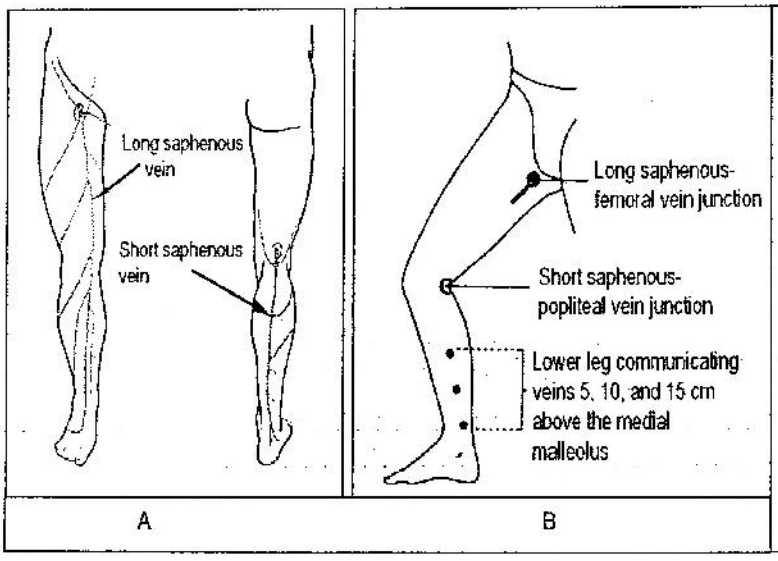

Long saphenous vein
Short saphenous vein
Long saphenous-femoral vein junction
Short saphenous-popliteal vein junction
Lower leg communicating veins 5, 10, and 15 cm above the medial malleolus
A
B

FIGURE 14 – 3. The course of the long and short saphenous veins (A) and the sets of the communicating veins (B)

FEGAN'S TEST

Mark the varicosities with a skin pen while the patient stands then the patient is asked to lie down and raise the affected limb and rest the heel. The candidate then palpates the line of marked varicosities carefully for gaps in the deep fascia through which perforating vein pass (FIGURE 14 – 4). These gaps are felt as circular openings with sharp edges and are localised at about 5, 10, and 15 cm above the medial malleolus and represent the first, second, and third communicating sets respectively (FIGURE 14 – 3). Positive Fegan's test indicates that the varicose veins fill from perforators.

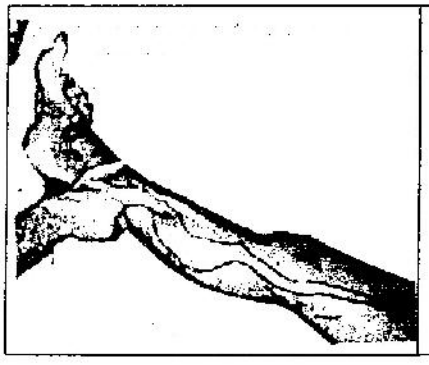


FIGURE 14 – 4. Fagan's test

AUSCULTATION

Listen over clusters of veins, particularly if they remain distended when the patient lies down. Bruits over these clusters of veins may indicate the presence of an arteriovenous fistula.

Complications of varicose veins are shown in BOX 14 – 3.

BOX 14 – 3. Complications of varicose veins	
Hemorrhage	Phlebitis
Ulceration	Oedema
Marjolin's ulcer	Dermatitis
Pigmentation	Cramps

EXAMINATION OF DEEP VENOUS THROMBOSIS

Venous thrombosis is divided into two types. These are:

1 Deep venous thrombosis

In which there is a thrombosis in normal deep veins. The vein wall may become inflamed as a secondary event, but often stays normal

2 Superficial venous thrombosis

In which, the vein wall is always inflamed and may be the cause of the thrombosis

Causes of deep and superficial vein thrombosis are shown in BOX 14 – 4.

BOX 14 – 4. Causes of deep and superficial veins thrombosis

Deep venous thrombosis	
Stasis from prolong immobility	High dosage estrogens
Post operative (> 30 minutes)	Intra-abdominal infections or tumour
Superficial venous thrombosis	
Varicose veins	Polycythaemia
Occult carcinoma e.g., pancreases, bronchus, and stomach	Polyarteritis nodosa
Buerger's disease	Idiopathic
	Iatrogenic due to intravenous injection or injuries

INSPECTION

Both legs must be exposed to look for superficial distended veins over the dorsum of the foot and over the upper third of the tibia on the affected side. Inspect for discoloration. Chronic venous insufficiency is associated with deposition of hemosidrin in the skin and the color may vary from deep blue/ black to purple or even bright red color. It is typically affecting the medial aspect of the lower third of the leg but the lateral aspect may also be affected. Inspect also for swelling and ulceration. A large swollen leg that is made pale by edema is called phlegmasia alba dolens (painful white leg). When the venous thrombosis blocks all the main veins, the skin becomes congested blue and the leg is called phlegmmasia cerulea dolens (painful blue leg).

PALPATION

Palpate (FIGURE 14 – 4) the instep and around the sulcus beneath the medial malleolus and the ankle for pitting oedema.

ROSE SIGN

Ascertain the presence of very early edema by pinching up a small portion of the skin where there is a resistance due to thickening of dermis and subcutaneous tissue.

ASSESS TEMPERATURE

Assess the temperature by the back of you fingers over the instep of both feet, shin of both legs and even both thigh and never forget to compare.

HOMAN'S TEST

Forceful dorsiflexion of the foot will cause pain in the calf (FIGURE 14 – 4). This test may cause dislodgement of the clot and consequently pulmonary embolism. Thus take a permission prior to performing this test

TENDERNESS

Try to find out tenderness in the following sites:

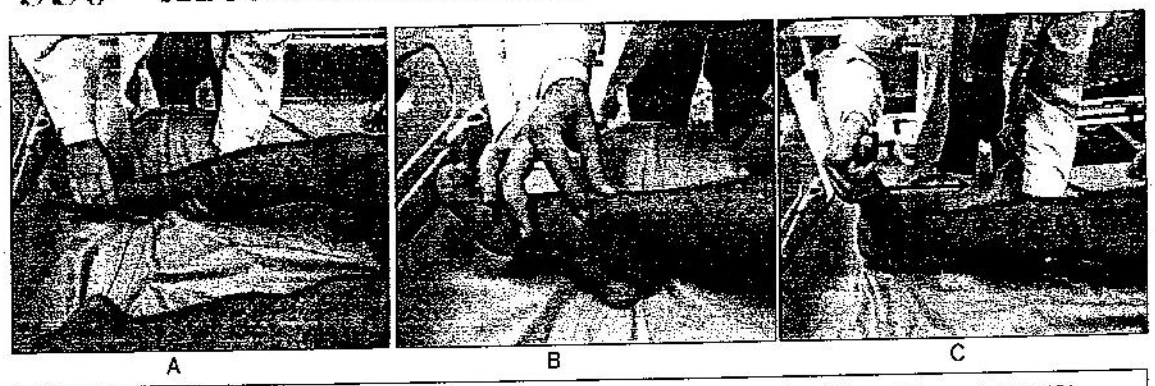
1 In the calves

Start grasping from the Achilles tendon in an upward direction till the soleus muscles to ascertain whether the soleus is tender. The tenderness elicited in this way is strongly suggestive of phlebothrombosis in posterior tibial vein. Do not forget the other calf muscle

2 In the popliteal space

3 In the thighs

Place the tip of the index finger over the saphenous opening and draw the finger down wards along the course of the femoral vein not along long saphenous vein



A B C

FIGUER 14 – 4. Palpation of the leg for the presence of thrombosis (A), Rose sign (B), and Homan's sign (C)

MEASUREMENT OF BOTH CALVES

Measure the claves in both legs at 10 cm from the tibial tuberosity; a difference of more than 3 cm is considered significant.

Differential diagnoses of a swollen leg are shown in BOX 14 – 5.

BOX 14 – 5. Differential diagnoses of leg swelling

Venous thrombosis	Baker's cyst (Popliteal cyst) usually occurs in combination with knee synovitis and the synovial fluid communicating with the cyst
Calf haematoma	
Skin inflammation including cellulites	Leg edema

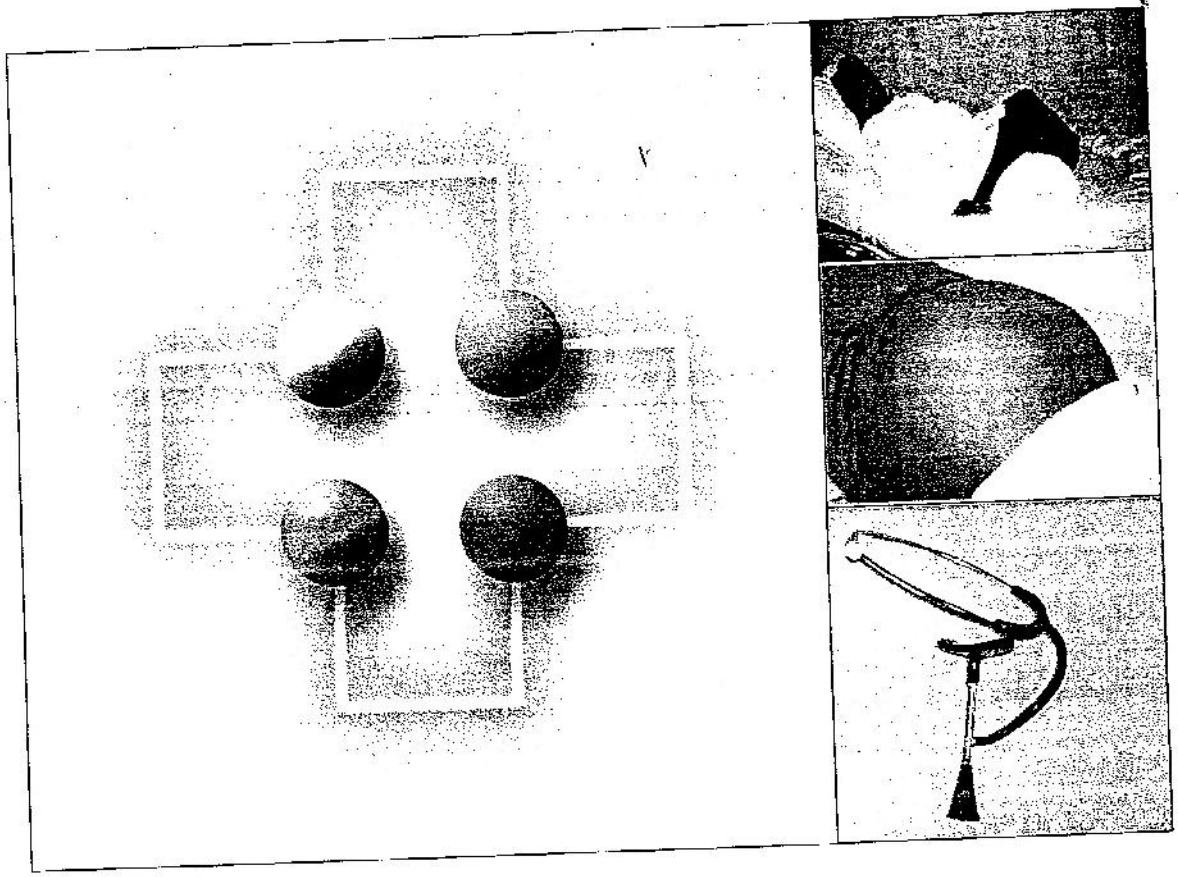
THE LYMPHATIC SYSTEM AND THE LYMPH NODES

The lymphatic system comprises an extensive vascular network that drains fluid, called lymph, from bodily tissues and returns it to the venous circulation. The system starts peripherally as blind lymphatic capillaries, and continues centrally as thin vascular vessels and then collecting ducts that finally empty into major veins at the root of the neck. The lymph transported in these channels is filtered through lymph nodes that are interposed along the way. Lymph nodes are round, oval, or bean shaped structures that vary in size according to their location. Some lymph nodes, such as the pre-auriculars, if palpable at all, are typically very small. The inguinal nodes, in contrast, are relatively larger often 1 cm in diameter and occasionally even 2 cm in adult.

In addition to its vascular functions, the lymphatic system plays an important role in the body's immune system. Cells within lymph nodes engulf cellular debris and bacteria and produce antibodies. Only the superficial lymph nodes are accessible to physical examination. These include the cervical nodes, the axillary nodes, and nodes in the arms and legs. Examination includes lymph nodes examination and lymphoedema (both had been discussed).

15

OBSTETRIC EXAMINATION



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INTRODUCTION

The techniques of history taking and examination for pregnant and non-pregnant women are similar. However, it is important to recognize the changes in anatomy and physiology that are related to pregnancy.
During pregnancy, there are changes in the thyroid gland, breasts, abdomen, and pelvis. You may also detect minor skin changes such as the mask of pregnancy and abdominal striae.
The process of birth varies greatly, depending on the relationships (lie, presentation, attitude, and position) of the fetus to the maternal anatomy. It is important to define these relationships accurately in order to follow the birth process.

1 LIE

The term lie refers to the relation of the long axis of the fetus to that of the mother. In more than 99% of full term pregnancies, the lie is in the same plane as or parallel to the long axis of the mother, a longitudinal lie. In rare instances, the long axis of the fetus is perpendicular to the maternal pelvis, a transverse lie

2 PRESENTATION

The term presentation refers to the part of the fetus in the lower pole of the uterus overlying the pelvic brim (e.g., cephalic, vertex, breech) and can be felt through the cervix. Usually the fetus's head is flexed so that the chin is in contact with the chest. In this case, the occipital fontanelle is the presenting part, and the presentation is referred to as a vertex presentation

3 ATTITUDE

The attitude (or habitus) of the fetus is the posture of the fetus: flexion, deflexion, or extension.
In most cases, the fetus becomes bent over so that the back is convex, the head is sharply flexed on the chest, the thighs are flexed over the abdomen, and the legs are bent at the knees. This is the description of the fetal attitude of flexion. Fetal postures are shown in FIGURE 15 – 1.

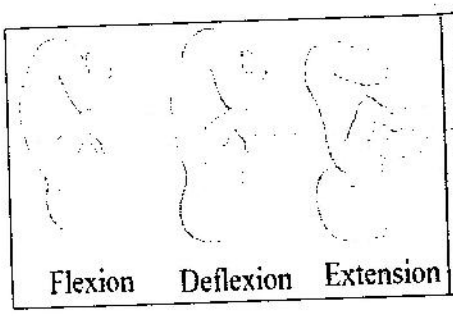
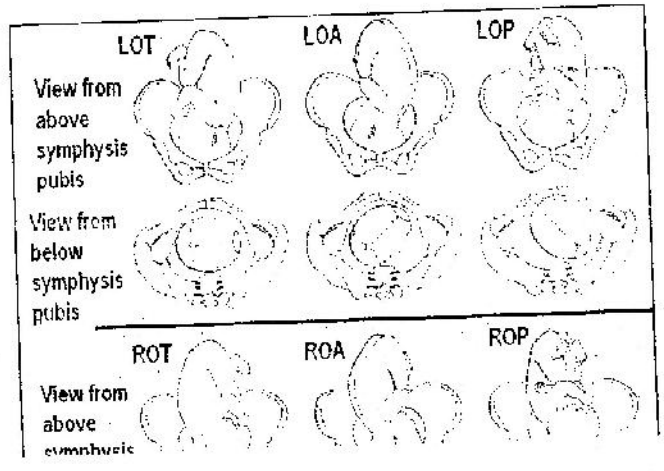


FIGURE 15 – 1. Fetal postures determining attitude

4 POSITION

The position is the relationship of an arbitrarily chosen portion of the presenting part of the fetus to the maternal pelvis. For example, in a vertex presentation, the chosen portion is the fetal occiput; in a breech presentation, it is the sacrum; and in a face presentation, it is the chin, termed the mentum. Because the arbitrarily chosen portion of the presenting part may be either left or right, the portion can be described as left occiput (LO), right occiput (RO), left sacral (LS), right sacral (RS), left mental (LM), or right mental (RM). This part is also directed anteriorly (A), posteriorly (P), or transversely (T). For each of the three presentations (vertex, breech, and face), there are therefore six varieties of position. For example, in a vertex presentation, if the occiput is in the left anterior segment of the maternal pelvis, the position is described as left occiput anterior (LOA). The common clinical vertex positions are illustrated in FIGURE 15 – 2. Left and right always denote the side of the mother. Likewise, anterior, posterior, and transverse refer to the mother's pelvis



5 STATION

The term station characterizes the level of descent of the presenting part of the fetus; "0 station" signifies that the fetal occiput has reached the level of the maternal ischial spines and that the widest transverse part of the baby's head (biparietal diameter) is at the level of the pelvic brim. This is also known as engagement. If the vertex is at –I station, it means that the fetal occiput is at a plane 1 cm above the level of the maternal ischial spines (and that the biparietal diameter is therefore 1 cm above the pelvic brim), and the baby's head is thus not engaged

There are four basic pelvic configurations: gynecoid, anthropoid, android, and platypelloid (FIGURE 15 – 3). These are based on the shape of the brim, mid-pelvis, and outlet. Any pelvis is likely to combine features of more than one configuration.

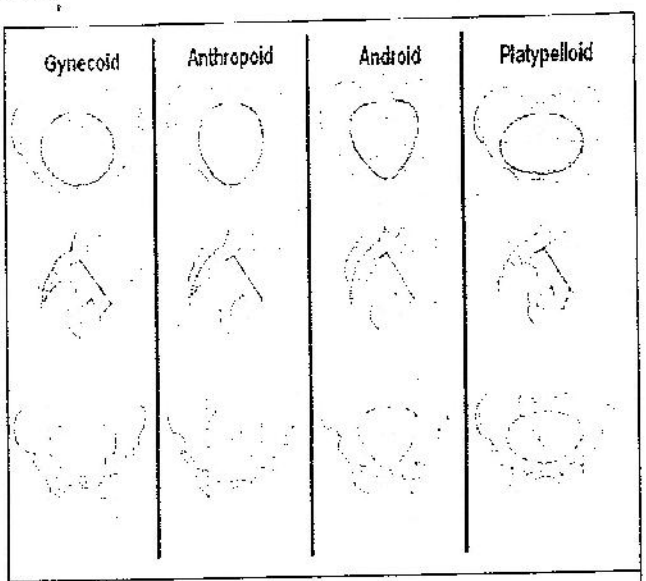


FIGURE 15 – 3. Basic pelvic configurations.

OBSTETRICAL AND GYNECOLOGICAL HISTORY

Obstetrical and gynecological history consists of the following elements:

PERSONAL HISTORY

The following data essential to be recorded from every patient admitted into the hospital:

1	Name	The patient and her husband
2	Age	The patient and her husband
3	Occupation	The patient and her husband
4	Address	The patient and her husband
5	ABO group and Rh group	The patient and her husband
6	Religion	The patient and her husband

7 Degree of relation between the patient and her husband
8 Next of kin
9 G (gravida) is the number of pregnancies regardless its outcome
10 P (para) is the number of deliveries after 28 weeks of gestation regardless a fetus live or dead
11 A (abortion) is the number of fetal loss before 24 weeks of gestation

EXAMPLES

(G_5 P_3 A_1) meaning a patient who had 1 abortion, 3 deliveries and now she is pregnant.
(G_4 $P^1{}_3$ A_0) meaning a patient who became 4 times pregnant, had 3 deliveries, but one of these deliveries resulted in a dead baby indicated by (1) and this baby may be still birth or intrauterine death (IUD) and should be died after the period designed for abortion and before labor.
(G_2 P_2 A_0) meaning twin pregnancy and she is pregnant.
(G_4 P_2 A_1 with positive history of H. mole) meaning a patient who had 2 deliveries with positive history of H. mole (ectopic pregnancy or H. mole) that is added to A (abortion) and now she is pregnant.

12 Last menstrual period (LMP)

Firstly, you have to ask about regularity of the period. If regular, ask about first day of the last menstrual period which is the day of flow.

13 Expected date of delivery (EDD)

The expected date of delivery can be calculated according to the last menstrual history (BOX 15 – 1).

BOX 15 – 1. Calculation of EDD	Days	Months	Years
L.M.P	+7	+9	Zero
In the first three months of the year	+7	3	+1
After the first three months			

An example for that is when the LMP on 20/10/2004, the EDD will be on 27/7/2005. Another example is when the LMP on 13/2/ 2005, EDD will be on 20/11/2005.

14 Gestational age or gestational weeks (GA)

Gestational age is calculated by the following two methods:

METHOD I Far from date (near LMP)

The duration between the LMP to the present date is calculated in weeks. Each two months is roughly equal to 9 weeks and each three months is roughly equal to 13 weeks. An example for that is when the LMP was on 6/ 2/2005 and the present date is 11/4/2005, so the duration is 2 months and GA is 9 weeks. Another example is when the LMP was on 1/ 2 / 2005 and the present date is 1/4/2005, so the duration is 2 months + 5 days and the GA is 9 weeks and 5 days.

METHOD II Near date (Far from LMP)

The duration between the present date and the EDD is calculated in weeks. Each two months is roughly equal to 9 weeks and each three months is roughly equal to 13 weeks. Then the result is subtracted from the full date in weeks. An example for that is when the present date is 1/4/2005 and EDD will be on 15/7/2005.
14 days means 2 weeks, and 3 months means 13 weeks. The result therefore is 15 weeks. Full date is 40 weeks. Thus the gestational week is equal to 40 weeks – 15 weeks = 25 weeks.
Note that date means 40 weeks and term is completed 37 weeks up to 42 weeks. Therefore full date is 40 weeks; postdate > 40 weeks: predate < 40 weeks. Full term is 42 weeks; post term is > 42 weeks: preterm means < completed 37 weeks.

15 Date of admission (DOA)
16 Date of history taken (DOH)

EXAMPLES

Mrs Zaineb Saddiq M. Aljubory; she is 16 years old, house wife, living in Diwaniya/Um alkheal, Moslem and of B+ve blood group. Her husband's name is Ahmed Ali M. Aljubory. He is twenty five years old, manual worker, Moslem and of unknown blood group. He is patient's cousin. The patient's next of kin is her mother. She is Para nill abortion nill ($G^1 P^0 A^0$) with no history of H. mole or ectopic pregnancy. Her LMP was on 15/12 /2004 thus EDD will be on 22/9/2005. Date of history taken is 13/3/2005 thus the gestational age is 17 weeks. The patient was admitted into the hospital on 8/3/ 2005.

Mrs Neveen Gorgies Basil Alma'aly. She is 25 years old, teacher, living in Baghdad/Alamil Quarter, Christian and of O +ve blood group. Her husband's name is Basil A. F. Al shamily. He is 32 years old, Engineer, Christian and of B – ve blood group.

He was not a relative to the patient. Neveen's next of kin is her sister. The patient is $G^1 P^2 A^1$ with positive history of H. mole. Her LMP was on 20/10/2004 thus EDD will be on 27/7/2005. DOH is on 16/2/2003 thus GA is 16 wks + 5 days.
The patient was admitted into the hospital on 15/2/2005.

CHIEF COMPLIANT AND ITS DURATION

Mention the main complaint (s) that brought the patient to the hospital with its duration. Again there is no way for medically correct terms. Examples include:

1 Vaginal bleeding for 2 hours duration prior to admission
2 Abdominal pain for 3 days duration prior to admission
3 Fever for 3 days prior to admission
4 Pallor of the skin for 1 month prior to admission

HISTORY OF PRESENT ILLNESS

As you learned earlier, this part of history consists of the following points:

1 Analysis of the chief complaint (s) in the form of patient's own wording
2 Analysis of the associated symptom (s)
3 Asking about the other symptoms of the involved system
4 Basic investigation that the patient had undergone meanwhile admission
5 Progression in the health status of the patient in the form of improvement or worsening since date of admission up to the time being now
6 In case of surgical history, mention the patient's condition post operatively in the form of fluid intake, medications, passing of flatus and movement of the patient

FEVER

Pregnancy does not alter the course of most specific fevers. High fever in pregnancy may lead to abortion or premature labor. Some organisms can reach the fetus and result in toxic effects e.g. rubella, small pox, chicken pox, typhoid fever, toxoplasmosis, and cytomegalovirus.

PRESENTATION: FEVER

1. Is the fever subjectively felt by the patient or it is genuine measured by a thermometer?
2. What is the mode of onset e.g. sudden or gradual?
3. What is the character of the fever e.g. continuous, intermittent, or remittent?
4. What are the relieving factors e.g. cool sponging or medications?
5. What are the associated symptoms e.g. sweating, chills, or rigor? If there sweating determine the time of occurrence (day or night time) and its nature whether profuse (drenching) or slight. Chills mean just bed shaking and are encountered in most febrile illnesses. However, a teeth-chattering, bed-shaking chill indicative of a true rigor is usually associated with bacteremia, pyelonephritis, abscess and biliary tract infection. Malaise, headache, and pain in the muscles and joints often accompany fever.
6. What are the timing of fever e.g. duration and course? Duration means the time since fever first noticed and the course means the progression of fever whether it started low grade then built up to be high grade or the fever was of low grade from the start.
7. What are the exacerbating and precipitating factors e.g. drugs or heat whether?
8. What is the severity of fever e.g. high, moderate or low grade? High grade fever made the patient unable to perform any activity, interfered with sleeping and eating and the patients are usually kept in bed.

CONVULSION IN PREGNANCY

Eclampsia is characterized by the occurrence of major epileptiform convulsions. Eclampsia remains one of the most serious complications of pregnancy. It may occur before, during or shortly after delivery.
Four stages of fits are described:

1 Orovisual phase — Flashes of light and spots before the eyes. The cry which is caused by spasm of the respiratory muscles and larynx (this is not usually noticeable)

2	Tonic phase	The patient loses her consciousness, has generalized muscular spasm and becomes cyanosed
3	Clonic phase	Violent movements occur, the tongue may be bitten and vomiting and inhalation of vomit may occur
4	In severe cases	Recurrent fits occur and the patient remains deeply unconscious between them

PRESENTATION OF SEIZURES: JERKING MOVEMENT OF LIMBS OR RIGHT SIDE...ETC

1. What are the events that preceded the convulsion e.g. the aura or simple partial seizure before secondary generalised tonic – clonic seizure and complex partial seizure or the aura before primary generalised tonic – clonic seizure?
2. What is the type of seizure e.g. partial (focal) or generalized fit?
3. What is the behavior of the patient during the seizure e.g. motor (tonic – clonic, clonic, tonic, atonic, myoclonic, or absence) or sensory?
4. What is the duration of the seizure?
5. What are the provoking factors? (BOX 15 – 2)

SEAL IN

BOX 15 – 2. Provocative factors for seizure	
Sleep deprivation	Flickering Lights, including TV and computer screens (primary generalised epilepsies only)
Physical and mental Exhaustion	Intercurrent INfections and metabolic disturbances
Alcohol (particularly withdrawal)	

6. What are the associated symptoms during the convulsion e.g. cyanosis, urination, defecation, tongue biting?
7. What are the events that follow the convulsion e.g. headache, confusion, myalgia, or amnesia?
8. Are there features of pseudo-seizures?

DYSPNEA IN PREGNANCY

Oxygen requirement is increased 20% and the over breathing leads to a fall in partial pressure of carbon dioxide. The low pressure of carbon dioxide gives rise to a sensation of dyspnoea together with elevation of the diaphragm. In late pregnancy, the dyspnoea diminishes due to engagement of the fetal head.

PRESENTATION: SHORTNESS OF BREATH

1. Is dyspnoea induced by certain position e.g. orthopnea, trepopnea, or platypnoea? If the patient is orthopnoeic ask him or her about the numbers of pillows does he/she use.
2. What is the onset of dyspnoea e.g. acute or chronic?
3. What is the course of dyspnoea e.g. progressive or intermittent?
4. What are the relieving factors e.g. inhalation, oxygen, lying supine, or standing? Patients with acute pulmonary edema usually prefer to be upright, while those with pulmonary embolism are often more comfortable lying flat and may faint (syncope) if made to sit upright.
5. What are the associated symptoms e.g. cough, hemoptysis, and wheeze or chest pain?
6. What is the timing of dyspnoea e.g. day time or night time, and duration? Breathlessness that wakes the patient from sleep is typical of asthma and left ventricular failure (paroxysmal nocturnal dyspnoea). Patients with asthma typically wake between 3 and 5 a.m. and have associated wheezing. Breathlessness that is worst on waking in the morning is more typical of chronic obstructive pulmonary disease (COPD) and may improve after coughing up sputum.
7. What are the exacerbating and/or precipitating factors e.g. exercise, drugs, smoke, or stormy whether? Patients with exercise-induced asthma may notice that their breathlessness continues to worsen for 5-10 minutes after stopping activity. If you suspect asthma, ask about exposure to allergens, smoke, perfumes, fumes, cold air or drugs (aspirin or NSAIDs can trigger breathlessness). Common allergens are house dust mite (shaking bedding, hoovering), animals (cats, dogs, horses) and grass pollens and tree pollens. Breathlessness which improves at the weekend or on holiday suggests occupational asthma or extrinsic allergic alveolitis.
8. What is the severity of dyspnoea? Ask about the effect of work, heavy exertion, walking up hills or walking with contemporaries. Severely breathless patients are dyspnoeic at rest and while walking around the house, getting washed, dressing, and eating or doing light housework. Use grading scales for breathlessness (BOX 15 – 3).

BOX 15 – 3. Severity of breathlessness: Medical Research Council (MRC) classification	
Grade 1	Breathless when hurrying on the level or walking up a slight hill
Grade 2	Breathless when walking with people of own age or on level ground
Grade 3	Has to stop because of breathlessness when walking on level ground at own pace

VOMITING IN PREGNANCY

Nausea is the subjective feeling of a need to vomit. Vomiting is the oral expulsion of gastrointestinal contents resulting from contractions of gut and thoracoabdominal wall musculature. Retching is a strong involuntary effort to vomit. Vomiting is contrasted with regurgitation, the effortless passage of gastric contents into the mouth. Rumination is repeated regurgitation of stomach contents, which may be rechewed and reswallowed. In contrast to vomiting, these phenomena often exhibit volitional control. Normal symptom of pregnancy is morning sickness. The features of morning sickness are summarized in BOX 15 – 4. If the vomiting is persistent and disturbs the patient's health, it is termed hyperemesis gravidarum. It is essential to exclude a serious underlying cause in severe and excessive vomiting.

BOX 15 – 4. Clinical features of morning sickness of pregnancy

It starts about sixth to twelfth weeks of pregnancy
It occurs soon after waking
It is often nausea and retching rather than vomiting, but vomiting is still accepted symptom
It is slight not persistent
It does not disturb the patient's health

PRESENTATION: VOMITING

1. What is the severity of vomiting? How frequent is the vomiting?
2. What is the onset of the vomiting? Drugs, toxins, and gastrointestinal infections commonly cause acute symptoms, whereas established illnesses evoke chronic complaints. Chronic nausea and/or vomiting with no other abdominal symptoms is usually due to a psychological cause.
3. What is the character of vomiting e.g. contents, color, projectile or effortless, and the amount of each vomitus? Emesis of undigested food is consistent with a Zenker's diverticulum or achalasia. Blood in vomiting (hematemesis) raises suspicion of an ulcer, malignancy, or Mallory-Weiss tear whereas bilious vomiting excludes gastric obstruction. Feculent emesis is noted with distal intestinal or colonic obstruction or the presence of a gastrocolic fistula. Projectile vomiting can be due to raised intracranial pressure or due to gastric-outflow obstruction. Large volumes of vomit suggest intestinal obstruction.
4. What are the relieving factors e.g. drugs?
5. What are the associated symptoms with vomiting e.g. abdominal pain, fever, vertigo, or headache? Does the vomiting preceded by nausea, retching, or giddiness? Many gastrointestinal conditions are associated with vomiting, but nausea and vomiting without pain is frequently non-gastrointestinal in origin. Relief of abdominal pain by emesis characterizes small-bowel obstruction whereas vomiting has no effect on pancreatitis or cholecystitis pain. Fevers suggest inflammation; an intracranial source is considered if there are headaches or visual field changes; and vertigo or tinnitus indicates labyrinthine disease.
6. What is the timing of vomiting e.g. duration and course? Does it occur at the early morning time? Early-morning vomiting is seen in psychological cause, pregnancy, alcohol dependence and metabolic disorders.
7. What are the exacerbating and the precipitating factors e.g. drugs or foods?
8. What is the severity of vomiting?

EDEMA IN PREGNANCY

The pregnant normally increases her extra cellular fluid (a part from that in the fetus, placenta and liquor amnii) by over than 2500 cc during the course of pregnancy. Edema is resulted from excessive fluid retention because osmotic equilibrium is maintained by the retention of sodium and from pressure of the uterus on the pelvic veins, or by associated varicose veins. The edema can usually be detected over the tibia, feet and ankles.

PRESENTATION: ANKLE OR LEG SWEELING

1. Where is the site of the swelling e.g. generalized, localized (ankle, scrotum, or face and hands)? Edema limited to one leg or to one or both arms is usually the result of venous and/or lymphatic obstruction. Edema resulting from hypoproteinemia and fluid overload is characteristically generalized.
2. What is the onset of the attack e.g. sudden or gradual?
3. What is the character of the swelling e.g. pruritic, painful, and/or dusky or red?
4. What are the relieving factors e.g. diuretics, sleeping, or resting?
5. What are the associated features? Local tenderness and warmth suggest inflammation. Local cyanosis may signify venous obstruction. In individuals who have had repeated episodes of prolonged edema, the skin over the involved areas may be thickened, indurated, and often red.
6. What is the timing of the swelling (duration and course)? Is it confined to the feet and ankles or does it extend to the shin, knee, thigh, or even the buttocks, genitalia, and anterior abdominal wall? Is there any evidence of abdominal swelling and ascites? Edema resulting from hypoproteinemia is especially evident in the very soft tissues of the eyelids (patient feels unable to fully open his eyes), face and hands (the ring on a finger fits more snugly than in the past) and tends to be most pronounced in the morning because of the recumbent posture

assumed during the night. Edema associated with heart failure, by contrast, tends to be more extensive in the legs (patients feel shoes getting tight or complaining of difficulty in putting on shoes) and to be accentuated in the evening, a feature also determined largely by posture.

7. What are the exacerbating factors e.g. drugs, walking, sleeping, or menstruation?
8. What is the severity of the swelling? Severity is assessed as to whether the swelling interferes with the patient's daily activities.

FREQUENCY OF MICTURATION

Frequency of micturition occurs during the first 12 weeks, when the enlarging uterus is still in the pelvic cavity and presses the bladder. It may also occur in the last month of pregnancy when the presenting part of the fetus is engaging in the pelvis. Normal signs and symptoms of pregnancy are shown in BOX 15 – 5.

BOX 15 – 5. Clinical features of pregnancy

Six maternal symptoms	Six maternal signs	Six fetal signs
Amenorrhea	Enlargement of the body of the uterus	Ballottement
Morning sickness	Softening of the uterus	Palpable fetal parts
Breast symptoms	Softening and blue discoloration of the cervix	Palpable fetal movement
Frequency of micturition	Progressive enlargement of the uterus	Visible fetal movement
Abdominal enlargement	Painless contractions	Fetal heart sounds
Quickening	Uterine souffle	Funic souffle

VAGINAL BLEEDING

Slight bleeding in the early weeks of pregnancy may prove to be completely normal, but such bleeding may be the first indicator of abortion or ectopic pregnancy. In later weeks of pregnancy, such bleeding may be due to placental separation or other causes (BOX 15 – 6)

BOX 15 – 6. Some causes of vaginal bleeding in pregnancy

Vaginal bleeding prior to 24 weeks of pregnancy	Vaginal bleeding after 24 weeks of pregnancy
Abortion	Abruptio placentae
Ectopic pregnancy	Placenta praevia
H. mole	Incidental haemorrhage
Cervical erosion	Vasa praevia
Cervical polypi	
Cervical carcinoma	

PRESENTATION: VAGINAL BLEEDING

1. Amount of blood whether slight or heavy? Heavy vaginal bleeding is associated with passage of clots.
2. How frequent is the bleeding?
3. Is there any previous attack?
4. What is the duration?
5. What are the precipitating and aggravating factors?
6. What are the relieving factors?
7. Ask about the conceptus status. If the gestational age is less than 20 weeks, ask about the fetal movement but if the gestational age is more than 20 week, ask about the uterine contraction as well as fetal movements.
8. What are the associated factors e.g. vomiting, headache?

PAIN IN PREGNANCY

Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage. Most common type of pain in obstetrics is lower abdominal pain radiated to the back as well as both lower limbs.

PRESENTATION: ABDOMINAL PAIN

1. Where is the site of the pain? Visceral abdominal pain due to distension of hollow organs, excessive smooth muscle contraction, or mesenteric traction is poorly localised pain while parietal abdominal pain and abdominal wall pain is well localised.
2. What is onset of the abdominal pain e.g. sudden or gradual? In a previously asymptomatic patient, the sudden onset of severe abdominal pain, rapidly progressing to become generalized and constant, suggests perforation of a hollow viscus, a ruptured abdominal aortic aneurysm or mesenteric infarction. Preceding constipation suggests colorectal cancer or diverticular disease as the cause of perforation and prior dyspepsia suggests peptic

ulceration. Co-existing peripheral vascular disease, hypertension, heart failure or atrial fibrillation may suggest a vascular disorder, e.g. aortic aneurysm or mesenteric ischemia. During the first hour or two after perforation, a 'silent interval' may occur when abdominal pain resolves transiently. The initial chemical peritonitis may subside before bacterial peritonitis becomes established.

3. What is the character (nature) of the pain? Constant pain means steady pain that usually arises from a solid organ, e.g. pancreatitis. Colicky pain means intermittent pain that lasts for a short period of time (seconds or minutes), eases off and then returns. It arises from hollow structures, e.g. small or large bowel obstruction. Biliary 'colic' are misnamed, as the pain is constant rather than colicky. Biliary colic is the term used for the pain associated with the temporary obstruction of the cystic or common bile duct by a stone usually migrating from the gall bladder. Biliary colic is misnamed as the pain rapidly increases to a peak intensity and persists over several hours (up to four hours) before gradually resolving. More protracted pain, particularly when lasting more than 4 hours and associated with fevers and rigors, suggests secondary complications such as cholecystitis, cholangitis or gallstone-related pancreatitis.Other characters include stapping, dull-ache, constricting (squeezing), pricking, tearing, and heaviness in nature.
4. What is the radiation of the pain i.e. shifted or referred pain (BOX 15 – 7)? The term shifting pain means perception of pain in new site whilst the initial site of pain disappeared. The term referred pain means extension of pain to another site whilst the initial site of pain persisted.

BOX 15 – 7. Examples of pain radiation

The pain map	Radiation	Cause
From right or left iliac fossa to the corresponding testis	Referred	Urologic problem
From epigastrium to the back	Referred	Peptic ulcer
From right hypochondrium to the right shoulder	Referred	Gall bladder problem
From epigastrium where it disappeared then it appears in the right iliac fossa	Shifting	Appendicitis

In appendicitis, pain is initially localized around the umbilicus (visceral pain) and spreads as the inflammatory response progresses to involve the right iliac fossa (parietal or somatic pain). If the appendix ruptures, generalized peritonitis may develop. Occasionally, a localized appendix abscess develops, with a palpable mass and localized pain in the right iliac fossa.

5. What are the associated symptoms e.g. nausea or vomiting or headache?
6. What is the timing of the pain (duration, course, progression, and pattern)? Change in the pattern of symptoms suggests either that the initial diagnosis was wrong, or that complications have developed. In acute small bowel obstruction, a change from typical intestinal colic to persistent pain with abdominal tenderness suggests intestinal ischemia, e.g. strangulated hernia, an indication for urgent surgical intervention.
7. What are the precipitating, aggravating, and relieving factors? Precipitating factors refer to the factors that brought the pain where initially there was no pain; aggravating factors refer the factors that render the pain more severe where the pain was initially mild; and relieving factors refer to the factors that ease the pain or off it.
8. What is the severity of the pain? The severity of the pain is assessed by how much the pain interferes with sleeping, eating, or daily physical activity.

VAGINAL DISCHARGE

The vaginal watery transudation that occurs normally through vaginal wall is increased during pregnancy. Mucus from hypertrophied cervical glands is added to the vaginal transudes and desquamated vaginal cells. An excess of vaginal discharge results from infection.

PRESENTATION: VAGINAL DISCHARGE

1. How much is the amount e.g. little or excessive?
2. What is the color e.g. white, yellow, green or red?
3. Was there any blood, pus or clot?
4. How many times per day?
5. What are the aggravating factors?
6. What is the progression of her illness?
7. What are the associated factors e.g., pruritis, abdominal pain or fever?
8. Ask about the conceptus condition.

PALLOR OF THE SKIN

Anemia is defined according to the WHO (World Health Organization) as hemoglobin level of less than 13 g/dL in adult men and less than 12 g/dL in adult women.

PRESENTATION: PALLOR OF THE SKIN OR GENERALISED WEAKNESS OR EXERTIONAL SOB

1. What is the course of anemia e.g. single or repetitive attacks?

2. Does the patient have known type of anaemia e.g. thalassemias or sickle? At which age? Thalassemia major becomes symptomatic as severe and progressive hemolytic anaemia during the second six months of life.

Hemoglobin is a protein specially adapted for gas transport to and from the lungs. It is composed of heme and globin. The metabolism of heme component (iron + biliverdin) had been discussed under the section of jaundice earlier. The globin component is a protein consists of four globin chains that are made from two alpha and two non-alpha chains. Adult hemoglobin consists of hemoglobin A ($\alpha\alpha/\beta\beta$, > 97%), hemoglobin F ($\alpha\alpha/\gamma\gamma$, 1%), and hemoglobin A2 ($\alpha\alpha/\delta\delta$, <2%). Alpha globin chains are produced by two genes on chromosome 16 (thus we have four genes) and beta globin chains by a single gene on chromosome 11 (thus we have just two genes); imbalance in the production of globin chains produces the thalassemias. Complete absence of beta chain is denoted by β^0 and marked reduction in synthesis is denoted by β^+. Thalassemias are therefore classified in the following way:

1 β/β^+	β-thalassemia minor (trait)
2 β/β^0 or β^+/β^+ or β^+/β^0	β-thalassemia intermediate
3 β^0/β^0	β-thalassemia major
4 - $\alpha/\alpha\alpha$ (one alpha gene is deleted)	α thalassemia carrier
5 - $\alpha/-\alpha$; - - $/\alpha\alpha$ (two alpha genes are deleted)	α thalassemia trait
6 - $\alpha/-$ - (three alpha genes are deleted)	Hemoglobin H disease
7 - -/- - (four alpha genes are deleted)	Hydrops fetalis

Amino acid substitutions in the beta chains will change the function of the globin chain at critical sites and result in clinical condition called hemoglobinopathies. Examples of hemoglobinopathies are shown here:.

1 β6 glutamic acid to valine	Hemoglobin S (sickle)
2 β6 glutamic acid to lysine	Hemoglobin C
3 β26 glutamic acid to lysine	Hemoglobin E

3. Has the patient have relevant medical or surgical history? Past medical history may reveal a disease which is known to be associated with anaemia, such as rheumatoid arthritis (the anaemia of chronic disease), or previous surgery (e.g. resection of the stomach or small bowel which may lead to malabsorption of iron and/or vitamin B_{12}).
4. Was it preceded by ingestion of a substance e.g. fava bean? G6PD is suspected in such a case.
5. Is there any family history of anemia? Hemolytic anemias such as the hemoglobinopathies and hereditary spherocytosis may be suspected from the family history. Pernicious anaemia may also be familial.
6. Was it preceded by ingestion of drugs? G6PD can follow intake of analgesics (aspirin), antimalarials (quinine, chloroquine), antibiotics (sulphonamides, ciprofloxacin), and other drugs (vitamin K, dapsone). Autoimmune hemolytic anemia may follow methyldopa, mefenamic acid, penicillin, quinine. Drugs can also be associated with blood loss (NSAIDS) or may cause aplasia of the bone marrow (chloramphenicol, sulphonamides).
7. Was there any blood transfusion? What is the number of blood pints received?
8. What are the associated factors e.g. tiredness, jaundice, gastrointestinal bleeding, or heavy menstruation? Iron deficiency anaemia is the most common type of anaemia world-wide. A thorough gastrointestinal history is important, looking in particular for symptoms indicating blood loss. Menorrhagia is a common cause of anaemia in females still menstruating, so women should always be asked about their periods.
9. How is the diet of the patient? A dietary history should assess the intake of iron and folate which may become deficient in comparison to needs e.g. in pregnancy or during periods of rapid growth.

CLINICAL CLUE

A clinical manifestation unique to iron deficiency is pica, which is an unusual craving for certain non-nutritional substances. Pica may be manifested as a craving for ice (pagophagia), or less commonly for clay (geophagia) or starch (amylophagia); pagophagia is believed to be the most specific to iron deficiency.

SYSTEMIC BLEEDING

Hemostasis depends upon interactions between the vessel wall, platelets and clotting factors. There are two phases of hemostasis: primary and secondary. In the primary phase, the damaged vessel constricts and platelets aggregate at the site of damage to form a plug to arrest hemorrhage within a few minutes. This is followed by activation of the coagulation system with secondary deposition of a fibrin mesh to secure the platelet plug. Bleeding results either from a breach of the vessel wall due to a specific insult (e.g. trauma) or from a defect in the hemostatic system. This may be due to a deficiency of one or more of the coagulation factors, thrombocytopenia, or occasionally excessive fibrinolysis which most commonly arises following therapeutic fibrinolytic therapy.

A history of bleeding is often remarkably reproducible, particularly after dental extraction; if a socket oozes for 2 days after removal of a tooth on one occasion; this is likely to recur following each subsequent extraction.

PRESENTATION: BLEEDING

1. Where is the site of bleeds? Muscle and joint bleeds indicate a coagulation defect, whereas purpura, petechiae, ecchymoses, prolonged bleeding from superficial cuts, epistaxis, gastrointestinal hemorrhage or menorrhagia indicates a platelet disorder, thrombocytopenia or von Willebrand disease.
2. Does the attack involve single or multiple sites? Bleeding at a single site suggest a local structural abnormality where as multiple sites bleeding suggests coagulopathy. However, even diffuse bleeding may arise from structural rather than hemostatic abnormalities (e.g., recurrent mucosal hemorrhage in patients with hereditary hemorrhagic telangiectasia). Conversely, a single episode of bleeding from an isolated site may be the initial manifestation of a systemic coagulopathy.
3. What is the duration of history? It may be possible to assess whether the disorder is congenital or acquired.
4. What are the precipitating causes? Bleeding arising spontaneously indicates a more severe defect than bleeding that occurs only after trauma.
5. Is there any relevant surgical history? Ask about all operations. Dental extractions, circumcision, tonsillectomy, labor and delivery, menses, vaccinations, and injections are particularly stressful tests of the haemostatic system. Bleeding that starts immediately after surgery indicates defective platelet plug formation and primary hemostasis, whereas that which comes on after several hours suggests a coagulation defect.
6. Is there any family history of bleeding? Absence of relatives with clinically significant bleeding does not exclude a hereditary bleeding diathesis; about one-third of cases of haemophilia arise in individuals without a family history.
7. Does the patient have any systemic illnesses? Many diseases (or their treatment) may be associated with bleeding but hepatic or renal failure, DIC, paraproteinemia or a connective tissue disease in particular should be considered.

HISTORY OF PRESENT PREGNANCY

Scientifically speaking, the pregnancy is divided into three trimesters (BOX 15 – 8).

BOX 15 – 8. The trimesters of the pregnancy

First trimester (1 – 13 weeks of gestation)

ASK ABOUT

Appetite. Normally it increases during pregnancy due to effect of progesterone
Nausea and vomiting (morning sickness)
Bowel habit e.g., constipation
Dysuria, color of urine, and backache
Early vaginal bleeding
Febrile condition

Antenatal care (ANC) means that pregnant women go to primary health centre (PHC) before and during pregnancy with the following aims:

- Maintain woman in health of body and mind.
- Anticipation of difficulties and complication of labor.
- Ensure birth of healthy infant.
- Help the mother rears the child.

Drug intake. The pregnant should not receive any drug during this period because it is a period of organogenesis.
Plain radiographs. radiation causes chromosomal anomalies
Ultrasonography

Second trimester (13 – 28 weeks of gestation)	Third trimester (28 weeks – full term)
ASK ABOUT	ASK ABOUT
Quickening which is the maternal perception for the first fetal movement. In primigravida, it occurs between 18 – 20 weeks while in multigravida, it occurs between 16 – 18 weeks Appetite, nausea, vomiting, and bowel habit Leg swelling (edema) Headache and visual disturbance Palpitation, dyspnea, and chest pain Paresthesia Weight gain Antenatal care (ANC). Vaccination (Tetanus toxoid)	Leg swelling Palpitation, dyspnoea, and chest pain Headache, visual disturbance, and fits Vaginal bleeding Abdominal pain

REVIEW OF OTHER SYSTEMS

1 Nervous system

Loss of consciousness, headache, convulsion, vision changes and paresthesia

2 Cardiovascular system

Chest pain, dyspnoea on exertion, palpitation, claudication, cyanosis and ankle oedema

3 Respiratory system

Chest pain, cough, sputum, haemoptysis, dyspnoea on exertion, and wheezing

4 Gastrointestinal tract

Appetite, nausea, vomiting, abdominal pain, heart burn, diarrhoea and constipation

5 Urological system

Frequency, polyuria, dysuria, nocturia, incontinence, change in color of urine, loin pain and thirst

6 Musculoskeletal system

Joint pain, backache and limitation of joint movement

7 Integumentary system

Skin rash, change in colour of urine and skin itching

PAST HISTORY

Past history consists of past medical history, past surgical history, past obstetrical history, and past gynecological history.

PAST MEDICAL HISTORY

Ask about the presence of previous medical illness including hypertension, diabetes mellitus, epilepsy, tuberculosis, rubella, rheumatic heart disease, and anaemia. Positive history should be analyzed with respect to its time, duration and treatment.

PAST SURGICAL HISTORY

Any type of surgical treatment the patient underwent e.g., appendicectomy, Cholecystectomy and nephroctomy...etc. here ask about time, indication, and complication if any.

PAST OBSTETRICAL HISTORY

Past obstetrical history should be analyzed in the form of:

1 Date of marriage

2 Any period of primary infertility. Infertility means no pregnancy after 1 year of unprotected coitus. If there is no pregnancy at all, it is termed primary infertility, but if there is a pregnancy then 1 year without pregnancy, it is called secondary infertility

3 History of each gravida as follows:

GRAVIDA ONE (G1)

- How pregnancy is diagnosed?
- Was it normal or abnormal pregnancy e.g. bleeding, infection, fit, vomiting or hypertension or diabetes mellitus?
- ANC; regular or poor
- Full term, preterm, post term.
- Onset of delivery; spontaneous or induced
- Site of delivery; at home or hospital.
- Mode of delivery.
- Outcome (baby), its sex, its outcome, its crying time, breast or bottle feeding, any congenital anomalies, and his status now.
- Peurperium is the period of returning menses which is normally 42 days. Ask about its complications as DVT, puerperal pyrexia, primary or secondary post partum haemorrhage, complication of breast feeding, and mental disorder

After that detect the period between G1 and G2; if it is long period ask about the cause whether secondary infertility or the use of contraception. Analyze G2 as G1, but if it is thoroughly normal, you can consider that the pregnancy is smooth and uneventful

PAST GYNECOLOGICAL HISTORY

This part of history should include analysis of the following five elements:

1. Past menstrual history. Ask about onset of menarche (first menstrual cycle that normally occurs between 11 – 14 years), regulation of the period, duration of every period, frequency of periods, dysmenorrhoea, and usual duration of each period and length of cycle, counting from the first day of one period to the first day of the next. This information is generally recorded as (M.C. 12 4/28 regular) meaning the periods began at the age of 12 last 4 days and occur every 28 days
2. Post coital bleeding that may suggest cervical carcinoma
3. Vaginal discharge (infection) or dyspareunia
4. Contraception and its type whether pills or IUCD
5. Any gynecological operation e.g., cuttery or colporrhaphy. Ask about time and type of the operation, indication, and complication

FAMILY HISTORY

Ask about cause of death of the patient's first degree relative e.g., parents, brothers and sisters, presence of hereditary disease e.g., diabetes mellitus or hypertension, and about history of twins, congenital anomalies or bleeding disorder.

DRUG AND HABITS HISTORY

Ask about chronic use of drugs, drug allergy/reaction, and habits in the form of alcohol intake, tobacco smoking and addiction.

SOCIOECONOMIC HISTORY

Ask about marital status of the patient e.g., married, separated, divorced or widow, type of home, size-owned or rented, rural or urban area, water supply (tap, well, river, or purified), and about indoor or outdoor domestic animals.

SPECIAL STUATIONS

ABORTION

Abortion is defined as termination of pregnancy before 24 weeks of gestation. WHO has defined abortion as expulsion or extraction from its mother of a fetus or an embryo weighing 500 grams. The clinical features of types of abortion are summarized in BOX 15 – 9.

BOX 15 – 9. Clinical features in abortion

Type of abortion	Uterine size	Cervix	Fetal heart
Threatened abortion	Normal	Closed	Present
Inevitable abortion	Normal	Open	Present or absent
Incomplete abortion	Reduced	Open	Absent
Complete abortion	Reduced	Closed	Absent
Missed abortion	Reduced	Closed	Absent

Abortion is classified into the following types:

1 Threatened abortion is characterized by slight vaginal bleeding, no or mild pain, viable fetus, and cervical os is closed
2 Inevitable abortion is characterized by vaginal bleeding, abdominal pain, and cervical os is dilated
3 Incomplete abortion means that part of the conceptus has been expelled, but there is continuous bleeding due to tissue retention
4 Complete abortion means that the whole conceptus has been expelled
5 Recurrent abortion means that three or more consecutive abortion
6 Missed abortion means that pregnancy failure is identified before expulsion of the fetus and placental tissue
7 Septic abortion results from ascending infection following illegal incomplete abortion

In this case ask about the following points:

1. Type of abortion.
2. The presenting symptom e.g. pain (ectopic pregnancy) or bleeding (abortion).
3. Other differential diagnoses of abortion e.g., ectopic pregnancy or H. mole.
4. Spontaneous or induced.
5. Gestational weeks.
6. Amount of blood lost.
7. Any infection precedes or follows abortion.
8. Blood transfusion received by the patient.
9. Evacuation which is better performed by dilatation and curettage (D and C) under general anesthesia.
10. Post abortive status.
11. Duration of staying in the hospital.
12. Rhesus group incompatibility.
13. Any previous similar condition.
14. Ask about the presence of risk factors:
    - Diabetes mellitus
    - Corpus lutium insufficiency
    - Cervical incompetence
    - Antiphospholipid antibody syndrome

CAESAREAN SECTION

Caesarean section is an operation by which a potentially viable fetus (> 28 weeks) is delivered through an incision in the abdominal wall and the uterus. Similar procedure undertaken to terminate pregnancy of a nonviable fetus (< 28 weeks) is referred to as hysterotomy. If such an incision is done in non pregnant women, it is termed as pfannestial incision.

In this case ask about the following points:

1. Type of caesarean; elective or emergency.
2. If elective, what are the predisposing measures?
3. If emergency, was there any trial of labor?
4. Indications of caesarean section (BOX 15 – 10).
5. Post operative measures; I.V. fluids and medications.
6. Post operative condition.

BOX 15 – 10. Indications of caesarean section

Absolute indications	Relative indications	X – Factors*
Marked cephalopelvic disproportion Transverse and oblique lie Placenta previa grade (II) posterior or grade (III) and (IV) Fetal distress in the first stage of labor Cord prolapse whenever vaginal delivery is not eminent Some malpositions as brow, face and in persistent mentoposterior Previous one upper segment caesarean section or more than one lower segment caesarean section Cervical carcinoma Herpes simplex type (II) 4 hours before rupture of membrane Pelvic tumour obstructing labor that is impacted in pelvis Severe preclampsia and eclampsia when vaginal delivery is not eminent	Intrauterine growth restriction Bad obstetric history as patient with unsuccessful pregnancy, abortion, neonatal death or intra uterine death	Breech presentation + previous scar. Breech presentation + severe preclamsia. Breech presentation + primigravida. Uncontroiled diabetes+ primigravida.

*If one factor is present, no indication for caesarean section. If more than one factor is present, cesarean section is indicated.

The condition of the patient postoperatively should be analyzed as follows:

Day zero
Complications of anesthesia
Recovery period
Post operative pain
Urinary catheter placement
Dysuria and chest pain
Vaginal bleeding
Breast feeding
Examination and medications

Day one
Mobilization
Removal of urinary catheter
Vaginal bleeding
Breast feeding
Examination and medications

Day two
Encouragement for mobilization
Febrile condition
Breast feeding
Examination and medications

Day three
Wound discharge
Any febrile condition
Hypercoagulable state
Breast feeding
Examination and medication

Day four onward
Removal of stitches (710 days)
Breast feeding
Examination and medication

Candidates should know that the history of present illness in case of cesarean section includes the analysis of the preceding questions. The chief complaint in elective caesarean section is patient with elective caesarean section. In case of emergency caesarean section on the other hand, labor pain is the main symptom that brought the patient to the hospital. Thus chief complaint is abdominal pain that should be analyzed as usual.

CLINICAL CLUE

In gynecological case, try not to ask about last menstrual period (LMP), expected date of delivery (EDD), gestational age (GA), and history of present pregnancy.

CASE PRESENTATION IN GYNECOLOGY

Mrs. Derwa Mohamed kadhem AlHashimy who is 55 years old, living in Najaf/Mushkab, Muslim, farmer, and of unknown blood group. Her husband's name is Alwan Yousif Yasir Al Hashimy. He is a second degree relative to patient. Derwa's next of kin is her grand daughter; Amal Muhammed Alwan. The patient is P6 A5, admitted into the hospital on 17/4/2002. Date of history taken is 19/4/2002.

CLINICAL CLUE

Note that there is no need to ask about gravida as the history is gynecological

She is chiefly complaining of vaginal discharge for three days prior to admission.

Regarding history of present illness. The condition started 6 months ago as a pain localized maximally to the back, of mild degree and of dull character. The pain gradually built up to be more severe particularly when the usual working of digging a ground was started. The pain also had lasted for few minutes radiated to the lower limbs, relieved only at the night when she lied flat and during sleeping then the pain disappeared to be recurred again and so on. The patient, however, had neglected her self because of the disappearance of pain.
After that, the patient felt a dragging sensation combined with a coming down swelling especially when she went to the bath as well as a difficulty in walking. Few days later the patient had developed frequent micturition and dripping of urine on coughing or sneezing. In addition, she experienced a difficulty in passing motions. Three days prior to admission, she started to have an excessive amount of green colored discharge associated infrequently with passage of blood. This complain occurred frequently along the day and associated with intermittent, high grade fever, thus the patient consulted a private clinic and admission was advised to her.
At the time of admission, urine sample and vaginal swab had been taken to lab, together with blood sample and CXR as well as an ultrasonography and an ECG had been performed (as a preparation for operation).
The patient received an I.V. fluid and injectable as well as oral form of medications. In addition, a urinary catheter with intermittent clamping is performed to her.
The patient now feels better after a decrease in her temperature and her vaginal discharge.

Regarding review of other systems.
Nervous system: no loss of consciousness, no convulsion and no vision changes.
Cardiovascular system: no chest pain, no dyspnoea, no palpitation and no cyanosis.
Respiratory system: no wheezing or haemoptysis.
Integumentary system: no skin rash. There was a redness of skin as well as skin itching at the internal region of each thigh.

Regarding past medical history:
The patient has a positive family history of tuberculosis (reactivation of primary tuberculosis as a case of chronic cough). This problem did appear since 2001 and a full course of oral tablets has been prescribed and administrated.

Regarding past surgical history.
The patient had operation in the form of appendicectomy 30 years ago with no complication encountered after it.

Regarding past obstetrical history.
The patient got married since 1967. Seven months later, she got a pregnancy diagnosed by the usual symptoms of amenorrhea and morning sickness. The patient had a poor ANC and vaginally delivered full term baby at her home (he is now aged 34 years, Amal's father). No complications had been encountered at that time in both the mother and the baby. Two years later, she got a pregnancy that unfortunately ended as abortion. The same picture had been repeated for the third gravida. The fourth, fifth and sixth gravidae had been completely passed smooth and uneventful. Two years later, she became a pregnant, but abortion was the dramatic result. One year after that, she got a pregnancy that passed uneventful and thoroughly smooth. The ninth and tenth gravidae ended as abortion. The last gravida resulted in a female sex out come.

Regarding gynecological history.
The patient had had a menopause 5 years ago. There was no use of contraception at the time of her fertility.

Regarding family history. The patient's partner had died 2 years ago due to CVA as well as both her parents. The patient is neither hypertensive nor diabetic. There is no family history of congenital anomalies of the uterus.
Regarding drug history. There is no chronic use on specific drugs. She had no allergy for penicillin.
Derwa is a chronic heavy smoker, smokes 20 cigarettes per day since 1980.

Regarding socioeconomic history. Derwa is living in a rural area. The river water is the usual water supply and there is indoor animales. Derwa had become unfortunately widow since 2000.

CASE PRESENTATION IN OBSTETRICS

Mrs. Zaineb Saddiq Malik Aljubory who is sixteen years old, house wife, living in Diwaniya/Um alkheal, Moslem and of B+ve blood group. Her husband's name is Ahmed Ali M. Al jubory, He is twenty five (25) years old, manual worker, Moslem and of unknown blood group. He is patient's cousin. The patient's next of kin is her mother and she is Para nill abortion nill (G1 P0 A0) with no history of H. mole or ectopic pregnancy.
Her LMP was on 15th /12/2004 thus EDD will be on 22nd /9/2005. Date of history taken is 15th /2/2005 thus the gestational age is 9 wk. The patient admitted into the hospital on 8th /2/ 2005.

Regarding chief complaint and its duration. Lower abdominal pain for one day prior to admission.

Regarding history of present illness.
The condition started as a vaginal bleeding 2 days prior to admission that occurred intermittently, of slight amount, did not associated with passage of clots. Each attack lasted few minutes to be recurred irregularly. The whole attacks are four in numbers along the two days prior to admission. One day prior to her admission, she developed lower abdominal pain maximally localized to her right lower outer region of the abdomen, started as a mild degree then gradually built up to be of higher degree that interfered with her daily activities as sleeping or eating, but not sharp enough to render her crying of colicky character, lasting about 5 – 10 minutes to be relieved by lying flat and precipitated by walking and

usual daily activities. The pain was radiated to the right shoulder and some time to the lower limbs and associated with a single attack of little amount, bitter tasted, yellow colored vomiting, of effortless in nature and preceded by retching. The patient also experienced a period of tiredness and dizziness along that day. She, therefore, consulted a private clinic and admission was advised to her. Meanwhile admission, she received intravenous fluid therapy and oral form of medication. A blood sample is drawn for investigation as well as an ultrasonography. The pain now persists, but the bleeding had ceased.

Regarding history of present pregnancy.
First trimester (only, since the gestational age is 13) increased appetite at the start of pregnancy. Nausea and vomiting is started as early as the 6th week after diagnosis, but ceased at the 11th week. Normal bowel habit, no dysuria, normal colour of urine, regular antenatal care. No drugs had been taken at this period as well as no radiation or trauma has the patient exposed for. No fever was encountered at this period. A single ultrasonography had been taken at the hospital.
No second or third trimester yet.

Regarding review of other systems.
Cardiovascular system: no chest pain, no dyspnoea, no palpitation and no cyanosis.
Respiratory system: no cough, no haemoptysis and no wheezing.
Urological system: no dysuria, no change in colour or odor of urine, but there was frequency.
Musculoskeletal system: no joint pain, no backache and no limitation of joint movement.
Integumentary system: no skin rash, no change in skin colour and no skin itching.

Negative past medical and past surgical history as well as past obstetrical history.

Regarding past gynecological history
M.C. 12 5/28 regular. The periods began at the age of 12, last for 5 days and occur every 28 days. No postcoital bleeding or dyspareunia. No use of contraception and no gynecological operation had been performed.

Regarding family history.
Both Zaineb`s parents are alive. No history of hereditary diseases as Diabetes Mellitus. No history of congenital anomalies and/or bleeding disorder.

Regarding drug history.
No chronic use on specific drug, no allergy to penicillin. Zaineb is neither smoker nor alcoholic.

Socioeconomic history.
Zaineb and her husband are living in an urban area together with the husband's family in a house supplied by tap water and proper sewage system and there is no domestic animals.

OBSTETRICAL EXAMINATION

The equipment necessary for examination of the pregnant woman is the same as for the non-pregnant woman. The patient's gown should open in the front for ease of examination. If the patient is in advanced pregnancy, avoid having her lie for a long period on her back, because the gravid uterus will diminish venous return and produce supine hypotension. It is useful for the woman to urinate before the pelvic examination. As always, wash your hands before beginning the examination. Make sure that your hands are warm and dry.
Because the examination of the pregnant woman is identical to the examinations described in the other chapters of this book, only special techniques and modifications for pregnancy are discussed in details in this part of the book.

VITAL SIGNS

I. BLOOD PRESSURE ASSESSMENT
In normal pregnancy, the systolic blood pressure is unchanged while the diastolic blood pressure is reduced at the first and second trimester, but then returned to non-pregnant level at term. When you examine the blood pressure, turn the patient to the left lateral decubitus to avoid compression of the uterus upon the inferior vena cava that results in a wrongly estimated hypotension.
II. BODY TEMPERATURE ASSESSMENT
III. RESPIRATORY RATE ASSESSMENT
IV. RADIAL PULSE ASSESSMENT
The pulse rate is increased by 8 – 16 beats per minute and the volume is increased normally during pregnancy by 3%. Due to this large volume of blood, a collapsing pulse is a usual finding.

GENERAL EXAMINATION

During pregnancy, there are changes in the thyroid gland, breasts, abdomen, and pelvis. You may also detect minor skin changes such as the mask of pregnancy and abdominal striae.
During pregnancy, the thyroid gland and the breasts undergo moderate enlargement due to hormonal stimulation and increased vascularity and hyperplasia of glandular tissue.
There may be tenderness and tingling in the breasts that makes them more sensitive during examination. By the third month of gestation, the breasts become more nodular, requiring careful palpation to avoid discomfort as you examine for any breast masses. The nipples become larger and more erectile. From mid to late pregnancy, colostrum, a thick, yellowish secretion rich in nutrients, may be expressed from the nipples. The areolae darken, and Montgomery's glands are more prominent. The venous pattern over the breasts becomes increasingly visible as pregnancy progresses. The abdomen's most notable change is distention, primarily from the increasing size of the growing uterus and fetus. Early distention from fluid retention and relaxation of abdominal muscles may be noted before the uterus becomes an abdominal organ (12 – 14 weeks of gestation). The expected growth patterns of the normal uterus and fetus and the standing contours of the primigravid abdomen in each trimester of pregnancy are illustrated in FIGURE 15 – 4.

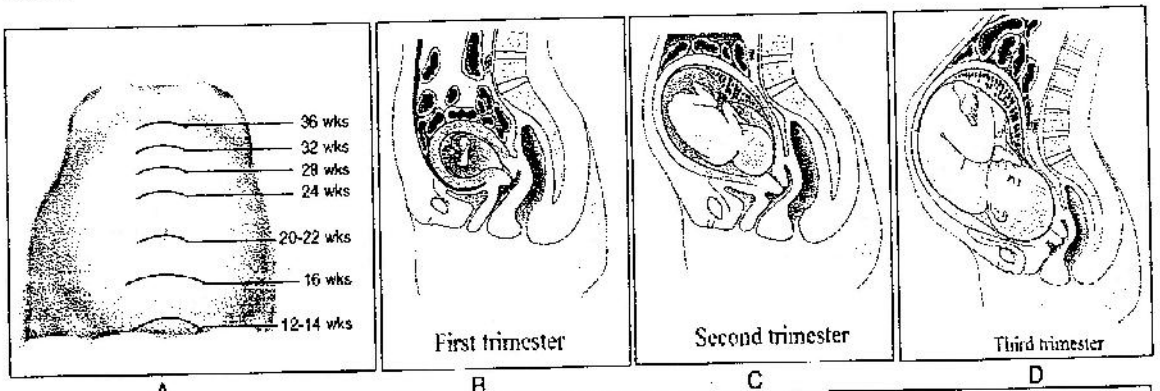


FIGURE 15 – 4. The expected growth pattern of uterus (A) and contours of the primigravid uterus (B, C, and D).

As the skin stretches to accommodate the growth of the fetus, purplish striae may appear. The linea nigra, a brownish black pigmented line following the midline of the abdomen, may become evident. Muscle tone is diminished as pregnancy advances, and diastasis recti, or separation of the rectus muscles at the midline of the abdomen, may be noticeable in the later trimesters of pregnancy. If diastasis is severe, as it may be in multiparous women, only a layer of skin, fascia, and peritoneum covers most of the anterior uterine wall. The fetus is felt easily through this muscular gap. The uterus is the organ most affected. Early in pregnancy, it loses the firmness and resistance of the non-pregnant organ. The palpable softening at the isthmus, called Hegar's sign, is an early diagnostic sign of pregnancy.

By 12 weeks' gestation, the uterus straightens and rises out of the pelvis and can be felt when palpating the abdomen. The enlarging uterus pushes the intestinal contents laterally and superiorly and stretches its supporting ligaments, sometimes causing pain in the lower quadrants. It adapts to fetal growth and positions and tends to rotate to the right to accommodate to the rectosigmoid structures in the left side of the pelvis.
The cervix also looks and feels quite different. Pronounced softening and cyanosis appear very early after conception and continue throughout pregnancy (Chadwick's sign). The cervical canal is filled with a tenacious mucous plug that protects the developing fetus from infection. Red, velvety mucosa around the os is common on the cervix during pregnancy and is considered normal.
The general examination is conducted through, as we have learned, two steps. The first step is the general look to the patient and the second step is regional examination. As you have learned earlier, a mnemonic (A SAD CBD) is used to describe the points of general look to the patient. These points are Age group, Sex (always female), Awareness, Decubitus (position of the patient in the bed), state of Comfortability, Body built, and Dyspnoea. In the regional examination, the approach starts with the examination hands and proceeds to the arms, head, neck, chest, abdomen, and lower extremities. We used to present any case taking particular consideration to Jaundice, Anemia (look for anemia in the conjunctivae and sublingual tissues not in the palms as pregnancy is associated with palmar erythema), Cyanosis, Clubbing, Oedema, and Lymphadenopathy. These signs are collectively known as JACCOL.

EXAMPLES

Young aged female, conscious, lying comfortable in Left lateral decubitus of normal body built, not dyspneic, not tachypneic. She had no pallor, jaundice, cyanosis, or clubbing, but mild bilateral pitting leg oedema, and no LAP. An I.V. cannula is inserted into her right arm.

Old aged female, drowsy, having a difficulty in her hearing, lying comfortable in a dorsal decubitus posture, obese, not dyspneic, not tachypneic, and severely pale. She had no jaundice no cyanosis, no clubbing, no oedema or L.A.P. and an I.V cannula inserted into her left arm.

THE HANDS
Palmar erythema is common during pregnancy. Another physiologic finding is large radial pulse volume and collapsing character.

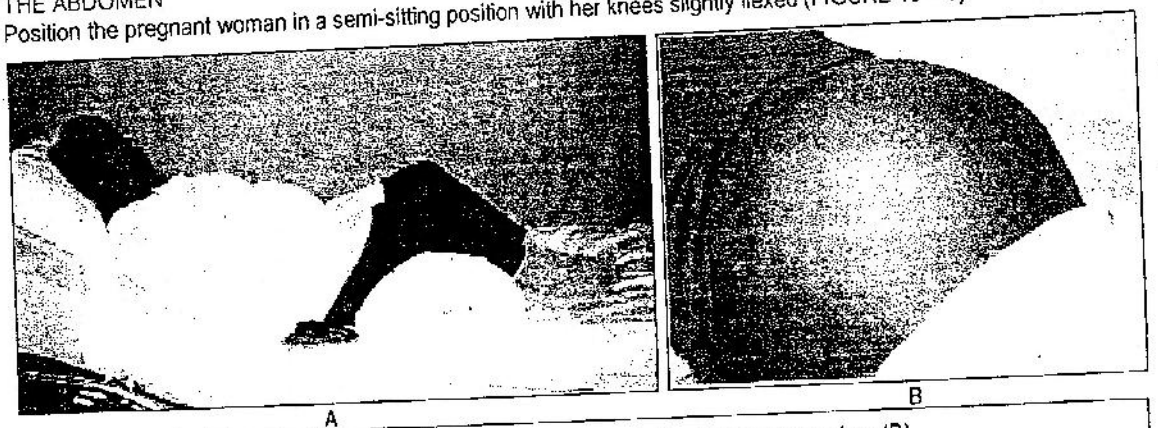
THE HEADS
The face during pregnancy becomes full, less wrinkles and rounded. Chloasma (mask of pregnancy) is irregular brownish patches on the checks due to increased secretion of melanocyte hormone. Facial edema after 24 weeks of gestation suggests pregnancy-induced hypertension. Dryness, oiliness, and sometimes minor generalized hair loss may be noted. Localized patches of hair loss should not be attributed to pregnancy. Anemia of pregnancy may cause pallor. Nasal congestion is common during pregnancy. Nosebleeds are more common during pregnancy. Gingival enlargement with bleeding is common during pregnancy.

THE NECK
Symmetric thyroid enlargement is expected. Marked or asymmetric enlargement is not due to pregnancy.

THE CHEST
Although women late in pregnancy sometimes report difficulty in breathing, there are usually no abnormal physical signs. If signs of respiratory distress are noted, examine the lungs thoroughly. Respiratory alkalosis is expected in later trimester. In advanced pregnancy, the apical impulse may be slightly higher than normal because of dextrorotation of the heart due to the higher diaphragm. Soft, blowing murmurs are common during pregnancy, reflecting increased blood flow in normal vessels. These murmurs may also accompany anemia.
Pregnant breasts become tender in early pregnancy and later on they will be enlarged due to fat deposition, growth of the alveoli and ductules. There will be primary and secondary areola. The nipples become enlarged, hypertrophied, hyperpigmented and erectile. Montgomery's tubercles are hypertrophied sebaceous glands look like rings present over the areola about 12- 20 small tubercles. The venous pattern may be marked, the nipples and areolae are dark, and Montgomery's glands are prominent. An inverted nipple needs an attention if breast feeding is planned.
Compression of the nipple between index finger and thumb may express colostrum from the nipples. A bloody or purulent discharge should not be attributed to pregnancy.

THE ABDOMEN

Position the pregnant woman in a semi-sitting position with her knees slightly flexed (FIGURE 15 – 5).



A B

FIGURE 15 – 5. Position of the patient during abdominal examination (A) and linea nigra (B).

INSPECTION

Inspection should include pattern of movement, contour of the abdomen, and the texture of the abdomen. The pattern of movement is normally thoracoabdominal i.e. mainly thoracic. If the occipit is anterior and the back of the fetus is to the front, the abdomen will appear smoothly convex; if the occiput is posterior, a flattening of the abdomen maybe seen above the symphysis pubis. If the lie is transverse, the uterus will be wider than usual and the height of the fundus may be little lower than with longitudinal lie. If twins are present, the uterus also appears wider, but the height of the fundus will be higher than normal. With these observations, you can decide whether the abdomen is symmetrically or asymmetrically distended. Generally, the umbilicus is displaced upward and it is flattened or even everted. Fetal movements can be noticed. Spider naevi may also be noticed due to increased estrogen level. Linea nigra (FIGURE 15 – 5) due to hyperpigmentation appears as a black line extends from the umbilicus downwards. Previous caesarean section scar is of value in the examination. Striae gravidarum are pink to red wrinkles due to stretching of the abdominal wall which lead to rupture of the elastic fibers. After delivery, these striae will be converted to white silvery striae known as striae albicantes.

PALPATION

Ask the patient about any tenderness and warm your hand. Generally the palpation is performed under three headings; superficial palpation, obstetric palpation, and deep palpation for visceromegaly.

I. Superficial (light) or general palpation

Place your hand's palmer surface flat and make counterclock wise direction around the boundaries of the uterus, searching for fetal movement, uterine contractility, superficial masses or tenderness.

Fetal movements can usually be felt by the examiner after 24 weeks. Quickening usually begins at 20 weeks in the primigravida but is felt 2 – 3 weeks earlier in the multipara. Although an important symptom, quickening is not a reliable sign of pregnancy because a woman can convince herself of its presence. If movements cannot be felt after 24 weeks, consider error in calculating gestation, fetal death or morbidity, or false pregnancy.

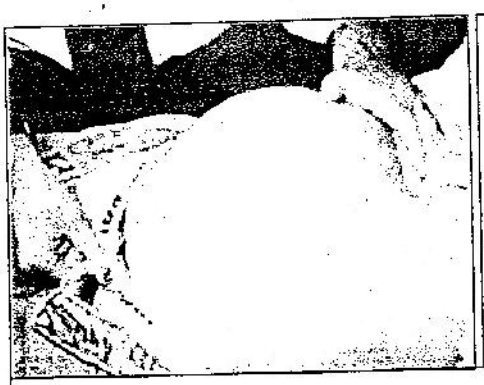
The uterus contracts irregularly after 12 weeks and often in response to palpation during the third trimester. The abdomen then feels tense or firm to the examiner and it is difficult to feel fetal parts. If the hand is left resting on the fundal portion of the uterus, the fingers will sense the relaxation of the uterine muscle. Prior to 37 weeks, regular uterine contractions with or without pain or bleeding are abnormal and suggest preterm labor.

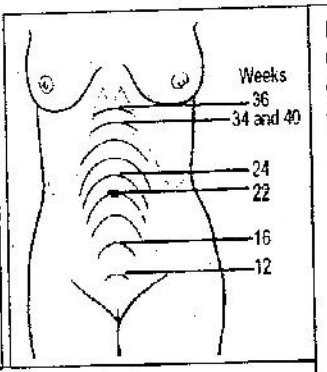
II. Obstetrical palpation.

These maneuvers are important adjuncts to palpation of the pregnant abdomen beginning at 28 weeks of gestation. They help to determine where the fetus is lying in relation to the woman's back (longitudinal or transverse), what end of the fetus is presenting at the pelvic inlet (head or buttocks), where the fetal back is located, how far the presenting part of the fetus has descended into the maternal pelvis, and the estimated weight of the fetus. This information is necessary to assess the adequacy of fetal growth and the probability of successful vaginal birth. These maneuvers are undertaken under five steps:

1. FUNDAL HEIGHT

Place the ulnar border of the left hand below the xiphysternum with the little finger moving it downwards. Measure the fundal height with a tape measure if the woman is more than 20 weeks' pregnant. Holding the tape as illustrated in FIGURE 15 – 6 and following the midline of the abdomen, measure from the top of the symphysis pubis to the top of the uterine fundus. After 20 weeks, measurement in centimeters should roughly equal the weeks of gestation. For estimating fetal height between 12 and 20 weeks, follow the expected fetal age shown in FIGURE 15 – 6. If the fetal age is more than 22 weeks, start from xiphysternum. But if the gestational age less than 22 weeks, start from umbilicus. If fundal height is more than 2 cm or less than 2 cm of the expected duration of pregnancy, consider the possibilities shown in BOX 15 – 11.





A B

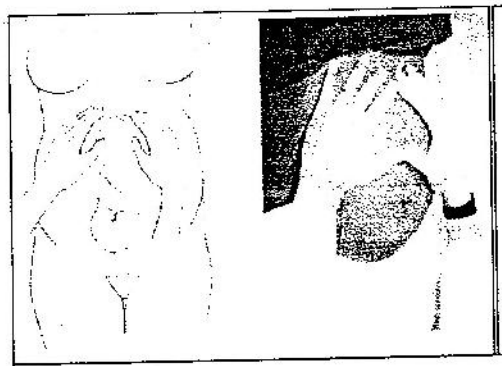
FIGURE 15 – 6. The technique used to estimate gestational age (A) and the corresponding gestational age (B).

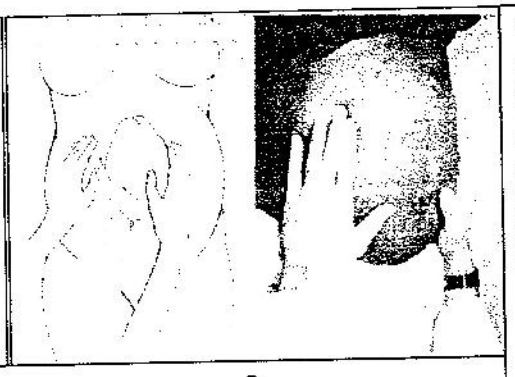
BOX 15 – 11. Expected duration of pregnancy

Fundal height higher than expected	Fundal height lower than expected
Multiple gestation	Missed abortion
A big baby	Transverse or oblique lie
Full bladder	Growth retardation or intrauterine death
Polyhydramnios	False pregnancy
H. mole	Abnormal fetus
Uterine myomata	Oligohydramnois
Error in date	Error in date

2. FUNDAL GRIP

It is also called the first Leopold's maneuver. It is used to evaluate the upper pole and defines the fetal part in the fundus of the uterus with both hands. This technique is demonstrated in FIGURE 15 – 7. Usually, the fetal buttocks are felt at the upper pole. They feel firm but irregular. The breech is continuous with the fetal back and a foot or knee can be felt near it and may move under the hands. If the head occupies the fundus, it is felt as rounder, harder, smother and more mobile mass than the breech. The head feels hard and round and is usually movable. The head can moved independently of the body and some times be balloted between the two hands.





A B

FIGURE 15 – 7. The technique of first Leopold's maneuver (A) and lateral grip (B)

3. LATERAL GRIP

It is also called the second Leopold's maneuver. The sides of the abdomen are palpated to discover on which side the back lies. If the back is directed more to the front than to the back a broad smooth surface will be felt on one side of the abdomen and on the other side, a number of small knobs, which are the limbs. If these small parts are felt all over the abdomen, the back must be directed posteriorly and to the side and deep palpation may be necessary to feel it. If the fetus is lying transversely, the head is felt in one flank and the breech is felt on the other side. If the fundus is lying obliquely, the head is often felt in one iliac fossa and the breech higher upon the opposite side. This technique is demonstrated in FIGURE 15 – 7.

4. FIRST PELVIC GRIP

As the majority of fetuses present by the vertex, the lower part of the uterus should be palpated to establish whether the presentation is cephalic. This is done by placing both hands on the lower abdomen above pelvic brim (FIGURE 15 – 8). The head is recognized because it is rounded and harder than the breech. Its mobility will depend upon its relation to the pelvic brim and as follows:

1 If it is completely above the pelvic brim, it is freely movable floating above the brim and the fingers of both hands can be made to meet below it. Thus engagement is not occur and the notation is 5/5 (five fifth palpable abdominally)
2 If it starts to enter the pelvic brim thus no engagement and the notation is 4/5 (four fifth palpable abdominally)
3 If the major part enter the brim thus no engagement and the notation is 3/5 (three fifth palpable abdominally)
4 If the widest part of the presenting part had been passed into the pelvic brim thus engagement occurs and the notation is 2/5 (two fifth palpable abdominally). The head here is usually fixed

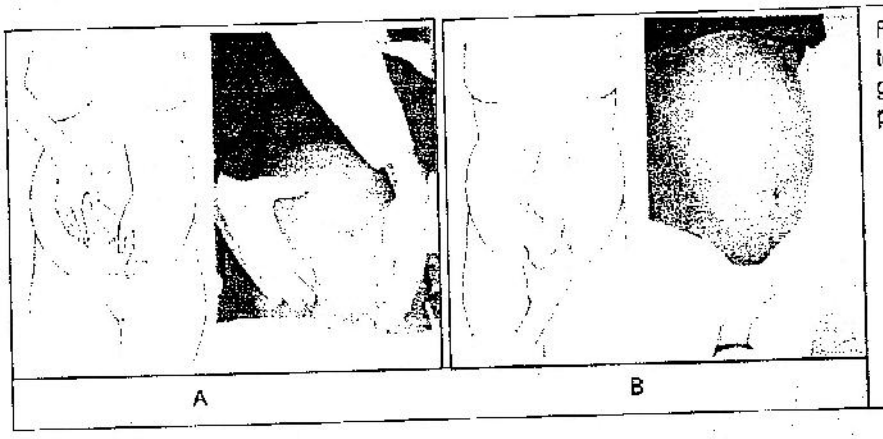


FIGURE 15 – 8. The technique of first pelvic grip (A) and second pelvic grip (B).

5. SECOND PELVIC GRIP (Pawlik's method)
By placing the right hand on the lower uterus and palpate using the thumb on one side and the fingers on opposite side to discover whether the presentation is cephalic or not and whether there is engagement (FIGURE 15 – 8).

III. Palpation of the abdominal viscera
The same techniques used for non pregnant are used here. However, in the third trimester palpation of the spleen is started from the left iliac fossa and palpation of the liver is start from the right iliac fossa or from the right hypochondrium.

PERCUSSION
The same techniques used for non pregnant are used here, but in addition, fluid thrill is positive in case of polyhydromnios.

AUSCULTATION
fetoscope called Pinard stethoscope (FIGURE 15 – 9) is used in age more or equal to 24 weeks of gestation to detect the fetal heart rate, rhythm and location. Lack of an audible fetal heart may indicate pregnancy of fewer weeks than expected, fetal demise, or false pregnancy. The rate is usually in the 160s during early pregnancy, and then slows to the 120s to 140s near term. After 32 to 34 weeks, the fetal heart rate (FHR) should increase with fetal movement.
An FHR that drops noticeably near term with fetal movement could indicate poor placental circulation. They are best heard over the chest or back of the fetus in vertex and breech presentation. In face presentation, they are heard over the front of the fetus. In occipitoposterior position (the back is directed to one side and posteriorly), they may be heard either in the flanks or near the midline. After 24 weeks, auscultation of more than one FHR with varying rates in different locations suggests more than one fetus.
Rhythm becomes important in the third trimester. Expect a variance of 10 to 15 beats per minute over 1 to 2 minutes. Lack of beat to beat variability late in pregnancy suggests fetal compromise.

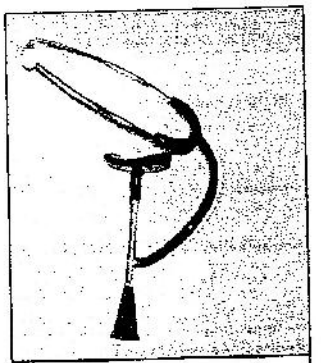


FIGURE 15 – 9. Pinard stethoscope.

GENITALIA, ANUS, AND RECTUM
Inspect the external genitalia, noting the hair distribution, the color, and any scars. Parous relaxation of the introitus and noticeable enlargement of the labia and clitoris are normal. Scars from an episiotomy, a perineal incision to facilitate delivery of an infant, or from perineal lacerations may be present in multiparous women. Some women have labial varicosities that become tortuous and painful. Varicosities often engorge later in pregnancy. They may be painful and bleed. Inspect the anus for hemorrhoids; note their size and location. Palpate Bartholin's and Skene's glands. No discharge or tenderness should be present. Check for a cystocele or rectocele.

SPECULUM EXAMINATION

Inspect the cervix for color, shape, and healed lacerations. A parous cervix may look irregular because of lacerations. A pink cervix suggests a non pregnant state. The cervix may bleed more easily if touched due to the vasocongestion of pregnancy. Vaginal infections are more common during pregnancy, and specimens may be needed for diagnosis. Inspect the vaginal wall for color, discharge, rugae, and relaxation. A bluish or violet color, deep rugae, and an increased milky white discharge, leukorrhea, are normal. A pink vagina suggests a non pregnant state. Vaginal irritation and itching with discharge suggest infection.

BIMANUAL EXAMINATION

Insert two lubricated fingers into the introitus (FIGURE 15 – 10) with slight pressure downward on the perineum. Slide the fingers into the posterior vaginal vault. Maintaining downward pressure, gently turn the fingers palmar side up. Avoid the sensitive urethral structures at all times. With the relaxation of pregnancy, the bimanual examination is usually easier to accomplish. Tissues are soft and the vaginal walls usually close in on the examining fingers, giving the sensation of being immersed in a bowl of oatmeal. It may be difficult to distinguish the cervix at first because of its softer texture. Place your finger gently in the os then sweep it around the surface of the cervix. A nulliparous cervix should be closed, while a multiparous cervix may admit a fingertip through the external os. The internal os, the narrow passage between the endocervical canal and the uterine cavity, should be closed in both situations. The surface of a normal multiparous cervix may feel irregular due to the healed lacerations from a previous birth. A shortened effaced cervix prior to 32 weeks may indicate preterm labor. Estimate the length of the cervix by palpating the lateral surface of the cervix from the cervical tip to the lateral fornix. Prior to 34 to 36 weeks, the cervix should retain its normal length of about 1.5 to 2 cm.

FIGURE 15 – 10.
Bimanual examination.

Palpate the uterus for size, shape, consistency, and position. These depend on the weeks of gestation. Early softening of the isthmus, Hegar's sign, is characteristic of pregnancy. The uterus is shaped like an inverted pear until 8 weeks, with slight enlargement in the fundal portion. The uterus becomes globular by 10 to 12 weeks. Anteflexion or retroflexxion is lost by 12 weeks, with the fundal portion measuring about 8 cm in diameter.

With your internal fingers placed at either side of the cervix, palmar surfaces upward, gently lift the uterus toward the abdominal hand. Capture the fundal portion of the uterus between your two hands and gently estimate uterine size. An irregularly shaped uterus suggests uterine myomata or a bicornuate uterus, which has two distinct uterine cavities separated by a septum.

Palpate the left and right adnexa. The corpus luteum may feel like a small nodule on the affected ovary during the first few weeks after conception. Late in pregnancy, adnexal masses may be difficult to feel.

Early in pregnancy, it is important to rule out a tubal (ectopic) pregnancy. Palpate for pelvic muscle strength as you withdraw your examining fingers. A rectovaginal examination may be done if you need to confirm uterine size or the integrity of the rectovaginal septum. A pregnancy of less than 10 weeks in a retroverted and retroflexed uterus lies totally in the posterior pelvis. Its size can be confirmed only by this examination.

EXAMINATION OF THE EXTREMITIES

General inspection may be done with the woman seated or lying on her left side. Inspect legs for varicose veins. Varicose veins may begin or worsen during pregnancy. Inspect the hands and legs for edema. Palpate for pretibial, ankle, and pedal edema. Physiologic edema is more common in advanced pregnancy, during hot weather, and in women who stand a lot. Pathologic edema associated with PET is often 3 + or more pretibially; it also affects the hands and face. Obtain knee and ankle reflexes. After 24 wk, reflexes greater than 2 + may indicate PET.

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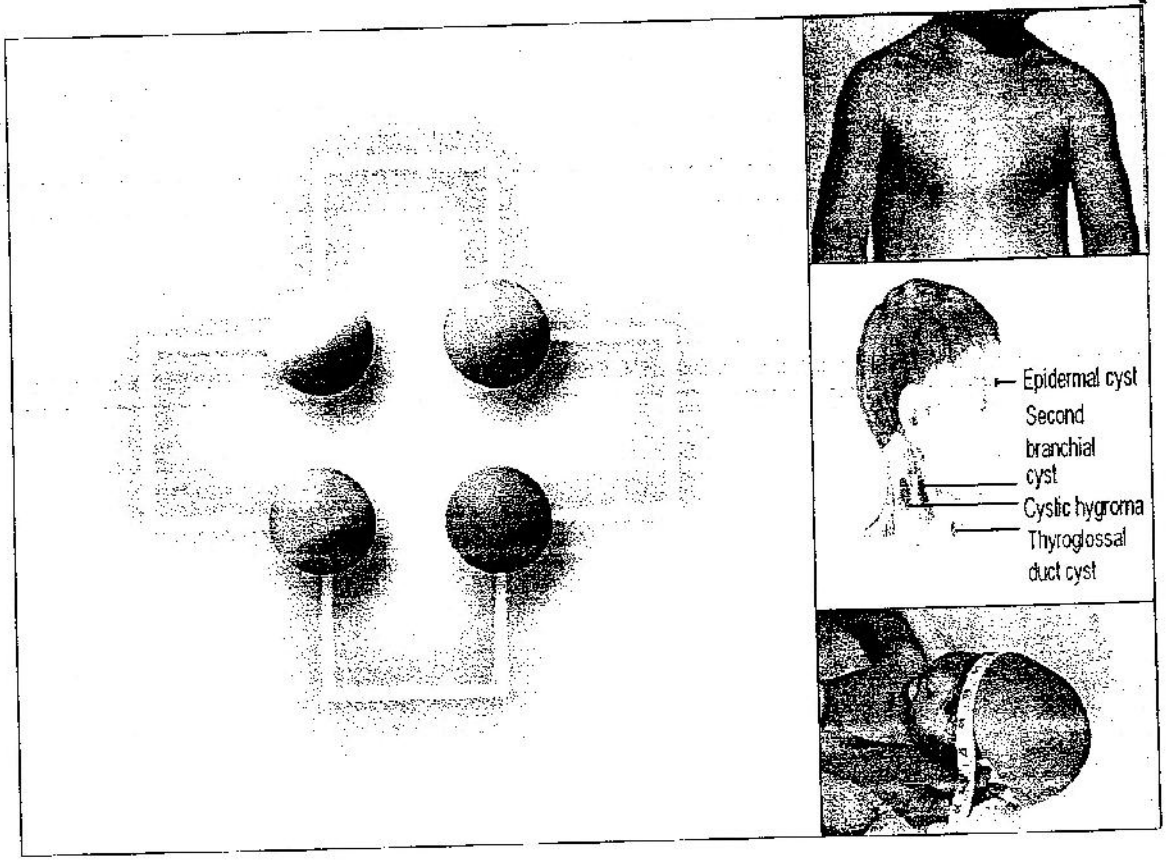
PEDIATRIC EXAMINATION



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INTRODUCTION

Children are anatomically and physiologically different from adults. Many techniques for assessment, physical findings, and even abnormalities in young patients differ as well. Infancy, the first year of life, is divided to the neonatal period (the first 28 days) and the postneonatal period (29 days to 1 year); early childhood is the period between 1 year through 4 years; middle childhood is the period between 5 through 10 years; adolescence is the period between 11 years through 20 years. This chapter is designed as two parts; the first part deals with the clinical history and clinical examinations of all pediatric patients apart from neonates. The second part deals with the clinical history and clinical examination of neonates. Both history and clinical examination have been modified to be suitably used in pediatric patients. The candidate should compare the information given with that given in early chapters as many clinical techniques have been modified from these used in adults. Similar techniques and clinical maneuvers have been not repeated here. The reader should, of course, read the details in earlier chapters of this book.

PEDIATRIC HISTORY

The pediatric history is quite different from adult history. In the latter the information of the history are thoroughly taken from the patient, but in the former the notes of the history are taken from the parents unless the child is in a suitable age. The history is best taken with the younger child in a comfortable position; usually on the mother's lap. The pediatric history consists of the following:

PERSONAL HISTORY

Leading questions are directed to the parents to identify the personal data of the child. These data include child's full name, age, sex, address, next of kin (usually the mother, but may be the grandmother or paternal or maternal aunt), and date of admission.

CHIEF COMPLAINT (S) AND ITS DURATION

Try to identify the main problem rendered the parents admitted their child into the hospital with its duration. As always, no way for medically correct terms.

HISTORY OF PRESENT ILLNESS

The same general outlines applied to adult history are now applied to pediatric history.

FEVER

Normal core body temperature is maintained at a set-point of 37°C, within a narrow range of 1 – 1.5° C. Elevation of body temperature above this range is termed fever. Consistently elevated body temperature of more than the set point for more than one week (2 – 3 weeks if an adolescent) for which basic examination and investigation for one week did not lead to a true underlying cause is called pyrexia of unknown origin (PUO).

PRESENTATION: FEVER

1. Is the fever subjectively felt by the child's parents or it is genuine measured by a thermometer?
2. What is the mode of onset e.g. sudden or gradual?
3. What is the character of the fever e.g. continuous, intermittent, or remittent?
4. What are the relieving factors e.g. cool sponging or medications?
5. What are the associated symptoms e.g. sweating, chills, or rigor? If there sweating determine the time of occurrence (day or night time) and its nature whether profuse (drenching) or slight. Chills mean just bed shaking and are encountered in most febrile illnesses. However, a teeth-chattering, bed-shaking chill indicative of a true rigor is usually associated with bacteremia, pyelonephritis, abscess and biliary tract infection. Malaise, headache, and pain in the muscles and joints often accompany fever.
6. What are the timing of fever e.g. duration and course? Duration means the time since fever first noticed and the course means the progression of fever whether it started low grade then built up to be high grade or the fever was of low grade from the start.
7. What are the exacerbating and precipitating factors e.g. drugs or heat whether?
8. What is the severity of fever e.g. high, moderate or low grade? High grade fever interferes with sleeping and eating and the child is usually kept in bed.

SEIZURES (CONVULSIONS)

Seizures are common neurologic disorder in the pediatric age group. Febrile seizures (BOX 16 – 1) are the most common seizure disorder during childhood with an excellent prognosis. However, febrile seizure may signify a serious underlying acute infection e.g., sepsis or meningitis.

PRESENTATION OF SEIZURES: JERKING MOVEMENT OF LIMBS OR RIGHT SIDE...ETC

1. What are the events preceded the convulsion e.g. the aura or simple partial seizure before secondary generalised tonic – clonic seizure and complex partial seizure or the aura before primary generalised tonic – clonic seizure?

BOX 16 – 1. Criteria for febrile seizure

Typical febrile seizure	Atypical febrile seizure
Develops when the core temperature is 39°C Generalized tonic clonic seizure Lasting 10 sec –10 min. Occurs once/day Affecting children 6 months– 6 yr Usually positive family history EEG is not warranted	Focal seizure Lasting > 15 minuts Repeated convulsions for several hours or days EEG is indicated

2. What is the type of seizure e.g. partial (focal) or generalized fit?
3. What is the behavior of the patient during the seizure e.g. motor (tonic – clonic, clonic, tonic, atonic, myoclonic, or absence) or sensory?
4. What is the duration of the seizure?
5. What are the provoking factors? (BOX 16 – 2)

SEAL IN

BOX 16 – 2. Provocative factors for seizure

Sleep deprivation Physical and mental Exhaustion Alcohol (particularly withdrawal)	Flickering Lights, including TV and computer screens (primary generalised epilepsies only) Intercurrent INfections and metabolic disturbances

6. What are the associated symptoms during the convulsion e.g. cyanosis, urination, defecation, tongue biting?
7. What are the events that follow the convulsion e.g. headache, confusion, myalgia, or amnesia?
8. Are there features of pseudo-seizures?

DYSPNEA

Dyspnoea is a common symptom of cardiac and respiratory diseases, but it may occur as a result of severe anaemia or diabetic ketoacidosis.

PRESENTATION: SHORTNESS OF BREATH

1. Is dyspnoea induced by certain position e.g. orthopnea, trepopnea, or platypnoea?
2. What is the onset of dyspnoea e.g. acute or chronic?
3. What is the course of dyspnoea e.g. progressive or intermittent?
4. What are the relieving factors e.g. inhalation, oxygen, lying supine, or standing?
5. What are the associated symptoms e.g. cough, hemoptysis, and wheeze or chest pain?
6. What is the timing of dyspnoea e.g. day time or night time, and duration? Breathlessness that wakes the patient from sleep is typical of asthma and left ventricular failure (paroxysmal nocturnal dyspnoea). Patients with asthma typically wake between 3 and 5 a.m. and have associated wheezing. Breathlessness that is worst on waking in the morning is more typical of chronic obstructive pulmonary disease (COPD) and may improve after coughing up sputum.
7. What are the exacerbating and/or precipitating factors e.g. exercise, drugs, smoke, or stormy whether? Patients with exercise-induced asthma may notice that their breathlessness continues to worsen for 5-10 minutes after stopping activity. If you suspect asthma; ask about exposure to allergens, smoke, perfumes, fumes, cold air or drugs (aspirin or NSAIDs can trigger breathlessness). Common allergens are house dust mite (shaking bedding, hoovering), animals (cats, dogs, horses) and grass pollens and tree pollens.
8. What is the severity of dyspnoea? Severe SOB interferes with the child's feeding and sleeping as well as renders the child cyanosed.

COUGH

Cough is the reflex response to the irritation of the lower respiratory tract or stimulation of the cough receptors in the airways mucosa. Asthma is the most common cause of cough in children (BOX 16 – 3).

BOX 16 – 3. Causes of cough in children

Reactive airways disease (asthma) Croup Bronchiolitis (first 2 years of life) Pneumonia Tuberculosis	Foreign body inhalation Pertussis Tracheitis Bronchitis Bronchiectasis, including cystic fibrosis Tracheomalacia

PRESENTATION: COUGH

1. Does the cough induce or increase in frequency by certain positions?

2. What is the onset of the cough e.g. acute, subacute, or chronic?
3. What is the character of the cough e.g. dry or productive, painful or painless? Does it occur in bouts suggestive of whooping cough? Productive cough in infants means a cough followed by vomiting. In a child with whoop, identify the post tussive emesis, contact with a similar case, and the vaccination schedule
4. What are the relieving factors e.g. drugs, hot shower?
5. What is the associated symptoms e.g. nasal discharge, fever, wheeze, or hoarseness?
6. What is the timing of the cough e.g. day time or night time, and duration?
7. What are the exacerbating and precipitating factors e.g. cold, dust or pollen?
8. What is the severity of the cough? How frequent is it?

SPUTUM PRODUCTION

Sputum production is common in patient with acute or chronic cough and its nature and appearance can provide valuable clues to the etiology.

PRESENTATION IS RELATED TO COUGH

1. What is the AMOUNT? Is it a small (a teaspoonful) or large (a teacupful) amount? Bronchiectasis causes large volumes of purulent sputum to be coughed up, varying with posture. Sudden production of large amounts of purulent sputum on a single occasion suggests rupture of a lung abscess or empyema into the bronchial tree.
2. What are the ASSOCIATED symptoms e.g. dyspnoea, fever, weight loss, or night sweat?
3. Is it ever BLOOD – stained?
4. At what POSITION is sputum most produced? In patients with bronchiectasis purulent sputum is varying with posture.
5. What is the COLOUR, TASTE, and SMELL of the sputum? Yellow sputum occurs in acute lower respiratory tract infection (due to the presence of live neutrophils) and in asthma (due to the presence eosinophils) while green sputum (due to dead neutrophils) indicates chronic infection in COPD or bronchiectasis. Purulent sputum is green because of lysed neutrophils which release the green-pigmented enzyme, verdoperoxidase. In early pneumococcal pneumonia sputum may be a rusty red colour, as pneumonic inflammation causes lysis of red cells. Chronic bronchitis and COPD cause clear or 'mucoid' sputum if there is no active infection while the first sputum produced in the morning by a patient with COPD may be green because of nocturnal stagnation of neutrophils. 'Foul' or 'vile'-tasting or -smelling sputum suggests anaerobic bacterial infection, and occurs in bronchiectasis, lung abscess and empyema. In bronchiectasis a change of sputum taste may indicate an infective exacerbation.
6. What is the CHARACTER of the sputum e.g. serous, mucoid, mucopurulent or purulent?
7. What is the DURATION of the attack?
8. At which TIME of the day does sputum is produced?

VOMITING

Nausea is the subjective feeling of a need to vomit. Vomiting is the oral expulsion of gastrointestinal contents resulting from contractions of gut and thoracoabdominal wall musculature. Retching is a strong involuntary effort to vomit. Vomiting is contrasted with regurgitation, the effortless passage of gastric contents into the mouth. Rumination is repeated regurgitation of stomach contents, which may be rechewed and reswallowed. In contrast to vomiting, these phenomena often exhibit volitional control. Some causes of vomiting are shown in BOX 16 – 4.

BOX 16 – 4: Causes of vomiting in children

Infants		Children	
Common	Rare	Common	Rare
Gastroenteritis Overfeeding Gastrooesophageal reflux Gastrointestinal obstruction Systemic infection	Inborn error of metabolism Brain tumor Food poisoning	Gastroenteritis Systemic infection Toxic ingestion Whooping cough Medications	Hepatitis Brain tumour Achalasia

PRESENTATION: VOMITING

1. What is the severity of vomiting? How frequent is the vomiting?
2. What is the onset of the vomiting? Drugs, toxins, and gastrointestinal infections commonly cause acute symptoms, whereas established illnesses evoke chronic complaints.
3. What is the character of vomiting e.g. contents, color, projectile or effortless, and the amount of each vomitus? Blood in vomiting (hematemesis) raises suspicion of an ulcer, malignancy, or Mallory-Weiss tear whereas bilious vomiting excludes gastric obstruction. Feculent emesis is noted with distal intestinal or colonic obstruction or the presence of a gastrocolic fistula. Projectile vomiting can be due to raised intracranial pressure or due to gastric-outflow obstruction. Large volumes of vomit suggest intestinal obstruction.

4. What are the relieving factors e.g. drugs?
5. What are the associated symptoms with vomiting e.g. abdominal pain, fever, or headache? Does the vomiting preceded by nausea, retching, or giddiness? Many gastrointestinal conditions are associated with vomiting, but nausea and vomiting without pain is frequently non-gastrointestinal in origin. Relief of abdominal pain by emesis characterizes small-bowel obstruction whereas vomiting has no effect on pancreatitis or cholecystitis pain. Fevers suggest inflammation; an intracranial source is considered if there are headaches or visual field changes; and vertigo or tinnitus indicates labyrinthine disease.
6. What is the timing of vomiting e.g. duration and course? Does it occur at the early morning time? Early-morning vomiting is seen in psychological cause, pregnancy, alcohol dependence and metabolic disorders.
7. What are the exacerbating and the precipitating factors e.g. drugs or foods?
8. What is the severity of vomiting?

DIARRHEA

Diarrhea is defined as an increase in the frequency, fluidity and volume of feces. World Health Organization has defined diarrhoea as a passage of more than three loose stools per day in a non-exclusively breast fed infants. Acute diarrhoea lasts less than 2 weeks but if persists more than two weeks, it is termed as chronic diarrhoea. Chronic diarrhoea is of two categories; secretory and osmotic. If the diarrhoea stops when medication or feeding discontinues, it is osmotic diarrhoea, but if persists despite discontinuation or in fasting patient, it is secretory diarrhoea. Common causes of diarrhoea are shown in BOX 16 – 5.

BOX 16 – 5. Causes of diarrhoea in children

Infectious causes		
Viruses	Bacteria	Parasites
Rotavirus Enteric adenovirus Norwalk-like viruses Coronavirus	Campylobacter jejuni Salmonella enteritides Shigella sonnei, flexneri,boydii and dysenteri Escherishia coli Yersinia enterocolitica Clostridium difficile Vibrio cholerae Staphylococcus aureus	Giardia lambilia Cryptosporidium Entamoeba histolytica

Non- infectious causes			
Feeding difficulty	Anatomical defects	Malabsorption	
	Hirschsprung's disease Short bowel syndrome	Cystic fibrosis Celiac disease	Fructose intolerance
Food poisoning	Neoplasms	Miscellaneous	
Heavy metals Mushrooms	Neuroblastoma Pheochromocytoma	Milk allergy Inflammatory bowel disease Haemolytic uremic syndrome	Protein losing enteropathy Laxative abuse

A clinical set of points to differentiate the underlying cause of diarrhoea is provided in BOX 16 - 6.

BOX 16 – 6. Differentiation of bacterial and viral gastroenteritis

Variable	Bacterial	Viral
Temperature > 38.5° C	Yes	Unusual
Abdominal pain and tenesmus	Yes	Unusual
> 8 bowel motions /day	Yes	Unusual
Emesis	Unusual	Yes
Duration > 5 days	Yes	No
Pus and mucus in the stool	Yes	No
Hematochezia	Yes	No, except rotavirus in preterm

PRESENTATION: FREQUENT BOWEL MOTIONS OR FREQUENT LOOSE STOOLS

1. What does the child's parent mean by diarrhoea? Diarrhoea can be defined by parents in a number of different ways; frequent stools, change in the consistency of the stools which have become loose or watery, fecal urgency (pseudodiarrhea), or fecal incontinence.
2. What is the mode of onset of diarrhoea e.g. acute or chronic? Acute diarrhoea means diarrhoea lasting less than 14 days and chronic diarrhoea lasting than 14 days?

3. What are the characters of the stools e.g. volume, shape (formed, unformed, or porridge –like, odor, and color (yellow, black, pale, green, white or silvery)? Secretory diarrhoea is likely if the diarrhoea is watery, painless, of high volume (commonly more than one liter per day) and persists when the patient fasts; there is no pus, mucus or blood, and the stools are not excessively fatty. Osmotic diarrhoea is characterised by its disappearance with fasting and by large volume stools related to the ingestion of food. Small bowel involvement is suggested when the bowel motion is of large volume, watery, not associated with blood but associated with abdominal bloating and cramping mid – abdominal pain while large bowel involvement is suggested when the bowel motion is of small volume and frequent and associated with blood and mucus in stool as well as cramping lower abdominal pain. Steatorrhoea is identified clinically when the stools are large volume, fatty or greasy, pale yellow to gray colored, extremely smelly, sometimes frothy and float in the toilet bowel (lavatory pan) and are difficult to flush away. Quantitatively, steatorrhea is defined as stool fat exceeding the normal 7 g/d; rapid-transit diarrhea may result in fecal fat up to 14 g/d; daily fecal fat averages 15–25 g with small intestinal diseases and is often >32 g with pancreatic exocrine insufficiency. Some patients pass small amounts of formed stool more than three times; the stools are not loose and stool volume is not increased. This is not true diarrhoea.
4. What are the relieving factors e.g. fasting, sleeping, or medications?
5. What are the associated symptoms e.g. local (mucus substance, pus, or blood) and systemic symptoms (abdominal cramps, tenesmus, fever, nausea and vomiting, or arthralgia? If there is blood with the stools does it mixed with or on the surface of the stool, or does it appear after passing the stool? Infectious diarrhoea is associated with the passage of pus with or without blood. Infectious diarrhea may be associated with systemic manifestations. Reiter's syndrome may accompany or follow infections by Salmonella, Shigella, Campylobacter, and Yersinia. Yersiniosis may also lead to an autoimmune-type thyroiditis, pericarditis, and glomerulonephritis. Both enterohemorrhagic E. coli (O157:H7) and Shigella can lead to the hemolytic-uremic syndrome with an attendant high mortality rate.
6. What is the timing of the diarrhoea (day or night time, course, and duration)? Diarrhoea occurring at night suggests organic disease.
7. What are the precipitating and/or exacerbating factors? Diarrhea closely following food consumption at a picnic, banquet, or restaurant may suggest infection with Salmonella, Campylobacter, or Shigella from chicken; enterohemorrhagic E. coli (O157:H7) from undercooked hamburger; Bacillus cereus from rice; Staphylococcus aureus or Salmonella from mayonnaise or creams; and Salmonella from eggs.
8. What is the frequency (number of motion/day) and severity of diarrhoea?

CONSTIPATION

Constipation is defined as infrequent passage of hard dry stools. Neonatal onset of constipation suggests Hirschsprung's disease. Constipation can be accompanied by pain due to hard large stools in the rectum thus more retention occurs and a vicious cycle ensues. Causes of constipation are shown in BOX 16 – 7.

BOX 16 – 7. Some important causes of constipation

Non-organic (functional)

Organic

Intestinal	Metabolic	Drugs	Neuromuscular
Hirschsprung's disease Stricture Volvulus Chaga's disease	Dehydration Cystic fibrosis Hypothyroidism Hypokalemia Renal tubular acidosis Hypercalcemia	Narcotic Antidepressants Vincristine	Myotonia dystrophica Spinal cord lesion

PRESENTATION: INABILITY TO PASS STOOLS OR PASSAGE OF HARD STOOLS

1. What does the child's parent mean by constipation? Constipation can refer to the passage of infrequent stools, hard stools or stools that are difficult to evacuate. Difficulty with evacuation of feces may occur with disorders of the pelvic floor muscles or nerves, or anorectal disease (e.g. fissure, or stricture). Patients with this problem may complain of straining, a feeling of anal blockage or even the need to self-digitate.
2. What is the mode of onset of constipation e.g. sudden or gradual?
3. What are the characters of the stool e.g. shape and color? Hard pellety stools occur with slow transit while loose watery stools are associated with rapid transit. Both small pellety or very large stools are more difficult to expel than normal stools.
4. What are the associated symptoms e.g. straining, abdominal cramps, blood, or diarrhoea? A chronic but erratic disturbance in defecation (typically alternating constipation and diarrhoea) associated with abdominal pain, in the absence of any structural or biochemical abnormality, is very common; such patients are classified as having the irritable bowel syndrome.
5. What is the timing of constipation (course and duration)?

6. What are the exacerbating and precipitating factors e.g. drugs, food, or stress?
7. What is the severity of the attack? Severity is assessed as whether the attack interferes with the patient's usual daily activities such as sleeping or eating.

CLINICAL CLUE

Anismus/dyschezia means difficulty emptying the rectum despite prolonged straining while tenesmus means persistent urge to empty the rectum with the feeling of incomplete evacuation.

ABDOMINAL PAIN

Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage. The pediatric patients are unable to communicate their pain experience clearly thus assessment of pain is necessarily indirect and includes the observation of cry, facial expression and autonomic responses and behaviors. The most valid indicator of pain in pediatric patients is facial expression. Some causes of abdominal pain are shown in BOX 16 – 8.

BOX 16 – 8. Some important causes of abdominal pain in children

Disease	Character	Disease	Character
Oesophageal reflux	Burning	Appendicitis	Sharp, steady
Duodenal ulcer	Sever burning, gnawing	Meckel's diverticulum	Sharp
Irritable bowel syndrome	Dull crampy	Intussusceptions	Crampy with pain free periods
Inflammatory bowel disease	Dull crampy	Pancreatitis	Constant, sharp
Intestinal obstruction	Alternating colic and pain free periods	Urolithiasis	Intermittent, sharp, and cramping
Lactose intolerance	Cramping	Urinary tract infection	Dull to sharp

PRESENTATION: ABDOMINAL PAIN

1. Where is the site of the pain? Visceral abdominal pain due to distension of hollow organs, excessive smooth muscle contraction, or mesenteric traction is poorly localised pain while parietal abdominal pain and abdominal wall pain is well localised.
2. What is onset of the abdominal pain e.g. sudden or gradual? In a previously asymptomatic patient, the sudden onset of severe abdominal pain, rapidly progressing to become generalized and constant, suggests perforation of a hollow viscus, a ruptured abdominal aortic aneurysm or mesenteric infarction. Preceding constipation suggests colorectal cancer or diverticular disease as the cause of perforation and prior dyspepsia suggests peptic ulceration. Co-existing peripheral vascular disease, hypertension, heart failure or atrial fibrillation may suggest a vascular disorder, e.g. aortic aneurysm or mesenteric ischemia. During the first hour or two after perforation, a 'silent interval' may occur when abdominal pain resolves transiently. The initial chemical peritonitis may subside before bacterial peritonitis becomes established.
3. What is the character (nature) of the pain? Constant pain means steady pain that usually arises from a solid organ, e.g. pancreatitis. Colicky pain means intermittent pain that lasts for a short period of time (seconds or minutes), eases off and then returns. It arises from hollow structures, e.g. small or large bowel obstruction. Biliary 'colic' are misnamed, as the pain is constant rather than colicky. Biliary colic is the term used for the pain associated with the temporary obstruction of the cystic or common bile duct by a stone usually migrating from the gall bladder. Biliary colic is misnamed as the pain rapidly increases to a peak intensity and persists over several hours (up to four hours) before gradually resolving. More protracted pain, particularly when lasting more than 4 hours and associated with fevers and rigors, suggests secondary complications such as cholecystitis, cholangitis or gallstone-related pancreatitis.

Other characters include stapping, dull-ache, constricting (squeezing), pricking, tearing, and heaviness in nature.

4. What is the radiation of the pain i.e. shifted or referred pain (BOX 16 – 9)? The term shifting pain means perception of pain in new site whilst the initial site of pain disappeared. The term referred pain means extension of pain to another site whilst the initial site of pain persisted.

BOX 16 – 9. Examples of pain radiation

The pain map	Radiation	Cause
From right or left iliac fossa to the corresponding testis	Referred	Urologic problem
From epigastrium to the back	Referred	Peptic ulcer
From right hypochondrium to the right shoulder	Referred	Gall bladder problem
From epigastrium where it disappeared then it appears in the right iliac fossa	Shifting	Appendicitis

In appendicitis, pain is initially localized around the umbilicus (visceral pain) and spreads as the inflammatory response progresses to involve the right iliac fossa (parietal or somatic pain). If the appendix ruptures, generalized peritonitis may develop. Occasionally, a localized appendix abscess develops, with a palpable mass and localized pain in the right iliac fossa.

5. What are the associated symptoms e.g. nausea or vomiting or headache?
6. What is the timing of the pain (duration, course, progression, and pattern)? Change in the pattern of symptoms suggests either that the initial diagnosis was wrong, or that complications have developed. In acute small bowel obstruction, a change from typical intestinal colic to persistent pain with abdominal tenderness suggests intestinal ischemia, e.g. strangulated hernia, an indication for urgent surgical intervention.
7. What are the precipitating, aggravating, and relieving factors? Precipitating factors refer to the factors that brought the pain where initially there was no pain; aggravating factors refer the factors that render the pain more severe where the pain was initially mild; and relieving factors refer to the factors that ease the pain or off it.
8. What is the severity of the pain? The severity of the pain is assessed by how much the pain interferes with sleeping, eating, or daily physical activity.

GASTROINTESTINAL BLEEDING

Hematemesis is defined as vomiting up blood. Melena is defined as a passage of black, tarry stools containing altered blood. Hematochezia is defined as a passage of red blood in the stools. When bleeding originates in the oesophagus, stomach or duodenum it causes haematemesis. Moderate to mild bleeding from sites above the distal ileum tends to cause melena while massive bleeding can cause hematochezia. Bleeding from sites beyond the distal ileum also cause hematochezia. Some causes of gastrointestinal bleeding in children are shown in BOX 16 – 10.

BOX 16 – 10. Some causes of gastrointestinal bleeding in children

Infants		Children	
Common	Rare	Common	Rare
Bacterial enteritis	Volvulus	Bacterial enteritis	Esophagitis
Milk protein allergy	Necrotizing enterocolitis	Peptic ulcer	Meckel's diverticulum
Intussusceptions	Haemorrhagic disease of	Intussusception	Foreign body
Swallowed maternal blood	newborn	Henoch-Schönlein purpura	Haemolytic uremic
Anal fissure	Meckel's diverticulum	Colonic polyps	syndrome
		Anal fissure	

PRESENTATION OF HEMATEMESIS: VOMITING OF BLOOD

1. Does it genuine hematemesis or hemoptysis? It is important to ensure the site from which the blood is coming. Thus it is important to ensure if vomiting of blood is reported, this is not the result of bleeding from a tooth socket or the nose, or coughing up of blood.
2. What is the onset i.e. does the attack comprise a sudden or gradual process?
3. What is the character e.g. fresh blood or coffee ground-stained fluid, in the first vomitus or subsequent vomitus? Hematemesis can be fresh and red when severe or degraded by gastric pepsin and becomes dark brown in colour and resembles coffee grounds when less severe. If the source of bleeding is above the gastro-oesophageal sphincter, e.g. from oesophageal varices, fresh blood may well up in the mouth, as well as being actively vomited. With a lower oesophageal mucosal tear due to the trauma of retching (Mallory-Weiss syndrome) the patient vomits forcefully several times and fresh blood only appears after the initial vomit.
4. What are the relieving factors e.g. drugs?
5. What are the associated features e.g. black tarry stools, weight loss, jaundice?
6. What is the timing of hematemesis (course and duration)?
7. What are the precipitating and the exacerbating factors e.g. alcohol, corticosteroid or NSAID therapy?
8. What is the severity of the attack (amount and the frequency)?

PRESENTATION OF MELENA: PASSAGE OF BLACK TARRY STOOLS

1. Where is the site of bleeding i.e. upper gastrointestinal bleeding or lower gastrointestinal bleeding? Fresh bleeding per rectum indicates hematochezia and in the absence of hemodynamic instability the likely source of bleeding is lower gastrointestinal region while black tarry stools indicates melena and the likely source of bleeding is upper gastrointestinal region while black tarry stools.
2. What is the onset of the attack e.g. sudden or gradual?
3. What is the character of the stools (tarry and shiny black or matt black stool) or the blood passed with it (mixed with the stools, on the surface of the stool, or appears after passing the stool)? Melena results mostly from upper gastrointestinal bleeding; the stool is 'tarry' and shiny black with a characteristic odor. Distinguish this from the matt black stools associated with oral iron or bismuth therapy. Hemorrhoids and local anorectal diseases such as fissures will commonly present with passing small amounts of bright red blood per rectum. The blood is not mixed in the stools but is on the toilet paper, on top of the stools or in the toilet bowl.

4. What are the relieving factors e.g. drugs?
5. What are the associated symptoms e.g. hematemesis, weight loss, or jaundice?
6. What is the timing of the attack (course and duration)?
7. What are the exacerbating and the precipitating factors e.g. alcohol, corticosteroid or NSAID therapy?
8. What is the severity of the attack (amount and the frequency)?

ANEMIA

It is defined as a reduction of the red cell volume or hemoglobin concentration below the range of values occurring in healthy per-sons. Pallor of the skin and mucus membrane is the usual manifestation and occurs together with tachycardia, shortness of breath, restlessness and irritability. Anaemia is not a disease entity but an indication of an underlying pathologic process or disease.

PRESENTATION: PALLOR OF THE SKIN OR GENERALISED WEAKNESS OR EXERTIONAL SOB

1. What is the course of anemia e.g. single or repetitive attacks?
2. Does the patient have known type of anaemia e.g. thalassemias or sickle? At which age? Thalassemia major becomes symptomatic as severe and progressive hemolytic anaemia during the second six months of life.

Hemoglobin is a protein specially adapted for gas transport to and from the lungs. It is composed of heme and globin. The metabolism of heme component (iron + biliverdin) had been discussed under the section of jaundice earlier. The globin component is a protein consists of four globin chains that are made from two alpha and two non-alpha chains. Adult hemoglobin consists of hemoglobin A ($\alpha\alpha/\beta\beta$, > 97%), hemoglobin F ($\alpha\alpha/\gamma\gamma$, 1%), and hemoglobin A2 ($\alpha\alpha/\delta\delta$, <2%). Alpha globin chains are produced by two genes on chromosome 16 (thus we have four genes) and beta globin chains by a single gene on chromosome 11 (thus we have just two genes); imbalance in the production of globin chains produces the thalassemias. Complete absence of beta chain is denoted by β^0 and marked reduction in synthesis is denoted by β^+. Thalassemias are therefore classified in the following way:

1 β/β^+	β-thalassemia minor (trait)
2 β/β^0 or β^+/β^+ or β^+/β^0	β-thalassemia intermediate
3 β^0/β^0	β-thalassemia major
4 - α/αα (one alpha gene is deleted)	α thalassemia carrier
5 - α/- α; - - /αα (two alpha genes are deleted)	α thalassemia trait
6 - α/- - (three alpha genes are deleted)	Hemoglobin H disease
7 - -/- - (four alpha genes are deleted)	Hydrops fetalis

Amino acid substitutions in the beta chains will change the function of the globin chain at critical sites and result in clinical condition called hemoglobinopathies. Examples of hemoglobinopathies are shown here:

1 β6 glutamic acid to valine	Hemoglobin S (sickle)
2 β6 glutamic acid to lysine	Hemoglobin C
3 β26 glutamic acid to lysine	Hemoglobin E

3. Has the patient have relevant medical or surgical history? Past medical history may reveal a disease which is known to be associated with anaemia, such as rheumatoid arthritis (the anaemia of chronic disease), or previous surgery (e.g. resection of the stomach or small bowel which may lead to malabsorption of iron and/or vitamin B_{12}).
4. Was it preceded by ingestion of a substance e.g. fava bean? G6PD is suspected in such a case.
5. Is there any family history of anemia? Hemolytic anemias such as the hemoglobinopathies and hereditary spherocytosis may be suspected from the family history. Pernicious anaemia may also be familial.
6. Was it preceded by ingestion of drugs? G6PD can follow intake of analgesics (aspirin), antimalarials (quinine, chloroquine), antibiotics (sulphonamides, ciprofloxacin), and other drugs (vitamin K, dapsone). Autoimmune hemolytic anemia may follow methyldopa, mefenamic acid, penicillin, quinine. Drugs can also be associated with blood loss (NSAIDS) or may cause aplasia of the bone marrow (chloramphenicol, sulphonamides).
7. Was there any blood transfusion? What is the number of blood pints received?
8. What are the associated factors e.g. tiredness, jaundice, gastrointestinal bleeding, or heavy menstruation? Iron deficiency anaemia is the most common type of anaemia world-wide. A thorough gastrointestinal history is important, looking in particular for symptoms indicating blood loss. Menorrhagia is a common cause of anaemia in females still menstruating, so women should always be asked about their periods.
9. How is the diet of the patient? A dietary history should assess the intake of iron and folate which may become deficient in comparison to needs e.g. in pregnancy or during periods of rapid growth.

A clinical manifestation unique to iron deficiency is pica, which is an unusual craving for certain non-nutritional substances. Pica may be manifested as a craving for ice (pagophagia), or less commonly for clay (geophagia) or starch (amylophagia); pagophagia is believed to be the most specific to iron deficiency.

SYSTEMIC BLEEDING

Hemostasis depends upon interactions between the vessel wall, platelets and clotting factors. There are two phases of hemostasis: primary and secondary. In the primary phase, the damaged vessel constricts and platelets aggregate at the site of damage to form a plug to arrest hemorrhage within a few minutes. This is followed by activation of the coagulation system with secondary deposition of a fibrin mesh to secure the platelet plug. Bleeding results either from a breach of the vessel wall due to a specific insult (e.g. trauma) or from a defect in the hemostatic system. This may be due to a deficiency of one or more of the coagulation factors or thrombocytopenia.

A history of bleeding is often remarkably reproducible, particularly after dental extraction; if a socket oozes for 2 days after removal of a tooth on one occasion; this is likely to recur following each subsequent extraction.

PRESENTATION: BLEEDING

1. Where is the site of bleeds? Muscle and joint bleeds indicate a coagulation defect, whereas purpura, petechiae, ecchymoses, prolonged bleeding from superficial cuts, epistaxis, gastrointestinal hemorrhage or menorrhagia indicates a platelet disorder, thrombocytopenia or von Willebrand disease.
2. Does the attack involve single or multiple sites? Bleeding at a single site suggest a local structural abnormality where as multiple sites bleeding suggests coagulopathy. However, even diffuse bleeding may arise from structural rather than hemostatic abnormalities (e.g., recurrent mucosal hemorrhage in patients with hereditary hemorrhagic telangiectasia). Conversely, a single episode of bleeding from an isolated site may be the initial manifestation of a systemic coagulopathy.
3. What is the duration of history? It may be possible to assess whether the disorder is congenital or acquired.
4. What are the precipitating causes? Bleeding arising spontaneously indicates a more severe defect than bleeding that occurs only after trauma.
5. Is there any relevant surgical history? Ask about all operations. Dental extractions, circumcision, tonsillectomy, menses, vaccinations, and injections are particularly stressful tests of the haemostatic system. Bleeding that starts immediately after surgery indicates defective platelet plug formation and primary hemostasis, whereas that which comes on after several hours suggests a coagulation defect.
6. Is there any family history of bleeding? Absence of relatives with clinically significant bleeding does not exclude a hereditary bleeding diathesis; about one-third of cases of haemophilia arise in individuals without a family history.
7. Does the patient have any systemic illnesses? Many diseases (or their treatment) may be associated with bleeding but hepatic or renal failure, DIC, paraproteinemia or a connective tissue disease in particular should be considered.
8. Does the patient have taken any drugs? Almost any medicine can potentially cause bleeding, either by depressing marrow functions with consequent thrombocytopenia or by interacting with warfarin e.g. NSAIDs inhibit platelet function, and the effect of aspirin may last for up to 10 days after a single tablet.

NEPHROTIC SYNDROME

Nephrotic syndrome is defined as a glomerular disease with a pentad of proteinuria, hypoalbuminemia, edema, hyperlipidemia, and lipiduria.

PRESENTATION: PUFFINESS OF THE FACE

1. What are the events preceded development of oedema?
2. Has the child had a recent or previous history of infection of the throat or skin? The typical patients develop an acute nephritic syndrome (post-streptococcal glomerulonephritis) 1 – 2 weeks after an antecedent streptococcal infection. Nephrotic syndrome may develop during the course of any type of glomerulonephritis.
3. Has the child had a history of chronic infection e.g., malaria and schistosomiasis? Nephrotic syndrome may develop during the course of chronic infection.
4. Has the child had a renal tumour?
5. Has the child had genital abnormalities? Drash syndrome consists of nephropathy, Wilm's tumour and genital abnormalities.
6. Has the child had congenital infection as syphilis, toxoplasmosis and cytomegalovirus? Nephrotic syndrome may develop in the first six months of life due to congenital infection.
7. Has the child had ingested drugs blamed for development of nephrotic syndrome? (BOX 16 – 11).

BOX 16 – 11. Drugs that may cause nephrosis

Penicillamine	Probenecid	Methimazole	Chlorpropamide	Trimethadione	Procainamide
Gold	Ethosuximide	Lithium	Phenytion	Paramethadione	Mercury

8. Has the child recently developed viral upper respiratory tract infection? The initial episode and subsequent relapses of nephrotic syndrome may follow an apparent viral upper respiratory tract infection.
9. How did the oedema progress? The edema is initially noted around the eyes and in the lower extremities then progressed to involve the abdomen, perineum and legs.
10. Ask about the gaining in weight, activity and appetite.

DIABETES MELLIYUS

Diabetes mellitus is defined as a clinical syndrome characterized by absolute or relative insulin deficiency with resistance to its metabolic action on the target cells leading to hyperglycemia and as a consequence micro-vascular and macrovascular disease, neuropathy, nephropathy, embryopathy, low resistance to infection and other short and long term complications. From the clinical point of view, the presentation is divided into three categories:

1 Those who have a history suggestive of diabetes, especially polyuria with polydepsia and failure to gain weight or a loss of weight in spite of a voracious appetite
2 Those who have a transient or persistent glycosuria
3 Those who have clinical manifestations of diabetic ketoacidosis with or without stupor or coma

Presentation of pediatric patients with diabetes mellitus is variable but they mainly have abdominal pain or loss of consciousness (in the third group), increased water intake and micturition (in the first and second group). Differential diagnoses are shown in BOX 16 – 12.

BOX 16 – 12. Differential diagnoses of diabetes mellitus

Urinary tract infection	Pneumonia	Salicylate intoxication
Diabetes insipidus	Acute abdomen	Any comatose child
Meningitis	Renal glucosuria	Gastroenteritis

1. How frequent is the micturition? Number of voiding per day.
2. Is there bed wetting in a previously toilet-trained child?
3. Is there loss of weight in spite of a voracious appetite.
4. Is there any type of infection? Pyogenic skin infection and monilial vaginitis in teenage girls due to low resistance to infection are occasionally present at the time of presentation.
5. Is there abdominal pain/abdominal distention?

JOINT PROBLEMS

Arthritis (BOX 16 – 13) implies the presence of synovial inflammation and includes the signs and symptoms of inflammation; swelling, loss of motion, pain, redness and warmth of the affected joint. Stiffness of the joint is an important additive indicator for joint disease. Arthralgia means pain in the joint with no other signs and symptoms.

BOX 16 – 13. Causes of arthritis in children

Infectious diseases	Vasculitis	Malignancy
Pyogenic arthritis Tuberculous arthritis Rheumatic fever Osteomyelitis Viral arthritis	Henoch-Schönlein purpura Kawasaki disease	Leukaemia Neuroblastoma Lymphoma Osteogenic sarcoma Other primary bone tumour
Non-inflammatory conditions	**Miscellaneous**	
Trauma Avascular necrosis Slipped capitofemoral epiphysis	Limb pain in children (growing pain) Sickle cell anaemia Hemophilia	Hypertrophic osteoarthropathy Juvenile rheumatoid arthritis (Still's disease)

PRESENTATION: LIMITATION OF JOINT MOVEMENT OR JOINT PAIN

1. Does the attack involve articular or non-articular structures?
2. What is the duration of the complain e.g. acute versus chronic? The duration of acute arthritis is less than 6 weeks while that of chronic arthritis is more than 6 weeks.
3. Does the joint complain inflammatory or non-inflammatory? With inflammatory joint complain the patients will usually have prolonged morning stiffness (more than 1 hour), local signs of inflammation (hotness, redness, tenderness, swelling, and loss of function), and systemic symptoms in the form of fever and weight loss.

CLINICAL CLUE

Pain belonging to the musculoskeletal system is divided into:

1. Usage pain. This pain is worse on use and relieved by rest. It indicates a mechanical strain.
2. Rest pain. This pain is worse after rest and improved by movement. It indicates an inflammation.
3. Night or 'bone' pain. This pain mostly occurs at night and poorly related to movement. It indicates a bone origin.

4. How many joints (BOX 16 – 14) involved in the attack e.g. monoarticular (one joint), oligoarticular or pauciarticular (2 – 4 joints) or polyarticular (more than 4 joints)?

BOX 16 – 14. Causes of arthritis according to the number of joints involved

Monoarthritis	Oligoarthritis	Polyarthritis
Septic arthritis Trauma Hemarthrosis in clotting abnormality Foreign body (e.g. plant thorn) Secondary avascular necrosis Monoarticular presentation of polyarthritis	Seronegative spondarthritis Infection as in bacterial endocarditis Juvenile idiopathic arthritis Oligoarticular presentation of polyarthritis	Rheumatoid arthritis Lupus arthritis Systemic sclerosis Polymyositis Viral arthritis Seronegative spondarthritis

5. When multiple joints are involved what is nature of involvement e.g. migratory or additive? Migratory means involvement of new joint and the previous one had been improved. Additive means involvement of new joint and the previous one still involved. Causes of migratory arthritis are shown in BOX 16 – 15.

BOX 16 – 15. Causes of migratory arthritis

Gonococcal arthritis	Relapsing polychondritis	Crohn's disease
Mumps arthritis	Leukemia	Whipple's disease
Acute rheumatic fever	Hyperlipidaemia	

6. Does the arthritis involve the axial or peripheral joints? If peripheral determine symmetry, small/large joints, predominance of upper/lower limbs (BOX 16 – 16).

BOX 16 – 16. Characteristic features of some polyarthritis

Cause	Characteristics
Rheumatoid arthritis	Symmetrical, small and large joints, upper and lower limbs
Seronegative arthritis	Asymmetrical, large > small joints, lower > upper limbs, spondylitis
Lupus	Symmetrical, small > large joints, joint damage uncommon
Systemic sclerosis and polymyositis	Rare, small and large joints
Juvenile idiopathic arthritis	Symmetrical, small and large joints, upper and lower limbs
Viral arthritis	Very acute, self-limiting

7. What are the relieving and the aggravating factors e.g. drugs?
8. What are the preceding events e.g. sexual contacts or dysentery? Reiter's disease or reactive arthritis typically develops 1-3 weeks following sexual exposure or an attack of dysentery.
9. Are there any extra-articular manifestations (BOX 16 – 17)?

BOX 16 – 17. Some extra-articular features of arthritis

Clinical feature	Disease association
Psoriasis, nail pitting and dystrophy	Psoriatic arthritis
Raynaud's phenomenon	Lupus, systemic sclerosis
Splinter haemorrhages, nail-fold infarcts	Vasculitis
Oral ulcers	Lupus, reactive arthritis, Behçet's syndrome
Large nodules (mainly extensor surfaces)	Rheumatoid arthritis, gout
Clubbing	Enteropathic arthritis, metastatic lung cancer, endocarditis
Uveitis	Seronegative spondarthritis
Episcleritis, scleritis	Rheumatoid arthritis, vasculitis
Pleuro-pericarditis	Lupus, rheumatoid arthritis
Fibrosing alveolitis	Rheumatoid arthritis, lupus, other connective tissue disease
Hepatosplenomegaly	Rheumatoid arthritis, lupus
Hematuria, proteinuria	Lupus, vasculitis, systemic sclerosis
Fever, lymphadenopathy	Infection, systemic juvenile idiopathic arthritis

JAUNDICE

It is defined as yellowish discoloration of the skin, sclera and mucous membrane. It is clinically evident if bilirubin exceeds 3 mg /dl. Jaundice in neonate is common and most cases are benign condition, however, severe untreated indirect hyperbilirubinemia may lead to death or survival with neurologic deficit. In addition, direct hyperbilirubinemia might indicate potentially serious illness. Immaturity of the blood brain barrier in neonates allows indirect bilirubin to pass to the brain and cause kernicterus or even death unlike in adults when pathology underlying the jaundice threatens the life not the jaundice. Classification of neonatal Jaundice includes:

1 Indirect hyperbilirubinemia:
   Non haemolytic
   Haemolytic:
      Extravascular hemolysis
      Intravascular hemolysis
2 Direct hyperbilirubinemia
3 Mixed hyperbilirubinemia

Direct hyperbilirubinemia (neonatal cholestasis) means prolong elevation of serum level of direct bilirubin beyond the first 14 days of life and generally more or equal to 20% of the total serum bilirubin e.g. total serum bilirubin 10 mg/dl and the direct fraction is 2mg/dl. According to the time of appearance, the causes of jaundice include:

1 Jaundice appears at birth or in the first 24 hours of neonatal life is pathologic and usually due to:
   Hemolytic disease of the newborn
   Concealed hemorrhage
   Congenital infection e.g., TORSCH (this acronym TORSCH refers to toxoplasmosis, other agents, rubella, cytomegalovirus, syphilis and herpes simplex)
2 Jaundice appears in the second and third day is due to:
   Physiological jaundice
   Early onset breast feeding
   Crigler-Najjar syndrome (rare)
3 Jaundice appears after the third day and within the first week is due to:
   Congenital infection
   Bacterial sepsis
   Extensive ecchymoses
4 Jaundice appears after the first week is usually prolonged jaundice and occurs due to:
   Late onset breast feeding
   Bacterial sepsis
   Biliary atresia
   Hepatitis
   Galactosemia
   Hypothyroidism
   Cystic fibrosis
   Congenital hemolytic anaemia

PRESENTATION: YELLOWISH DISCOLORATION OF THE SKIN, SCLERA, AND MUCUS MEMBRANE

1. What is the time of appearance of jaundice?
2. Is there family history of G6PD or hereditary spherocytosis?
3. What is ABO group for both mother and father?
4. What is Rhesus group for both mother and father?
5. Is there history of fever and rash in pregnancy that may suggest rubella or cytomegalovirus?
6. Is there any history of forceps delivery? This may suggest concealed hemorrhage.
7. What is the type of feeding e.g. breast or artificial bottle?
8. Is there any history of contact with a case of hepatitis?
9. What is the color of the stool, skin, and urine?

In older children, use the same axes of questions applied for adults.

LEARNING POINTS IN THE HISTORY OF PRESENT ILLNESS

1. In every symptom you have analyzed, ask the parents about feeding, appetite, weight gain or weigh loss, activities of the baby, and sleep disturbances.
2. A child with repeated admissions, ask the parents about the frequency of admissions, the length of each admission, the interval between each admission and another, and the improvement in the child's condition.
3. Drugs taken should be analyzed via their scientific and trade names, formulations; capsules, tablets, syrups, vials or ampoules, doses per day or per kg, rout of administration, I.V., I.M. or oral, and frequency of administration. It can sometimes be analyzed as simple as oral or injectable forms of medications.
4. Medical terms e.g. diarrhea and dyspnoea should be discouraged and instead use frequent bowel motions, shortness of breath (SOB) unless the word is popular as well as medical e.g. cough, vomiting, headache, or fever since these words are the patient's own wording.
5. Try not to mention names of doctors, instead use the following terms that are regarded as a source of referral:-
   - Private clinic.
   - Inpatient clinic.
   - Primary health center.
   - Hospital.
6. Any prior diagnosis in the history of present illness should not be recorded whether an informant said that or a patient's doctor unless the patient is a known case for that disease e.g. diabetes mellitus, hypertension, epilepsy and bleeding tendency and the current history is belonging to it. Here, the history of present illness is started by the following sentence (the patient is a known case of..... since .....). An example for that is the following case: a patient who had asthma for 3 years duration, 2 days earlier, he developed SOB. Thus the history of the present illness will be like this: the patient is a known case of asthma since 2010, two days prior to admission he developed SOB that was.....
7. The patient's complain should thoroughly be analyzed together with the associated symptoms. The remaining symptoms of the system involved should also be analyzed but to a lesser extent. An example for that is when a patient presented with abdominal pain. This pain should be thoroughly analyzed then the associated symptom(s) e.g. vomiting is analyzed. Lastly the related remaining symptoms of the gastrointestinal tract e.g. constipation because the patient's complain was one of the gastrointestinal symptoms.
8. If the patient for example suffered from headache for 3 days prior to admission, but developed abdominal pain for 1 day later that brought him/her to the hospital. Thus the chief complaint is abdominal pain for 2 days prior to admission, but the history of present illness will be as follow (the condition started as headache that was ....., one day after that the patient suffered from abdominal pain that was .....).
9. If the chief complaint for example is chest pain or dyspnoea so the systems involved are the cardiovascular and respiratory systems that should analyzed in the history of present illness and not mention in the next step of history which is review of other systems.
10. Patients with metabolic disease e.g. chronic renal failure when develop decompensated event they usually present with shortness of breath (SOB) due to fluid overload. The systems involved are thus the respiratory and the cardiovascular systems. In such exceptional case or any similar case one should in addition analyze the genuine underlying system which is the urolological system looking particular at the urine volume, color, and form or edema in the history of present illness. A similar case involved the hematological system when a patient presented with shortness of breath (SOB) due to anemia.
11. During analysis of the case, you may use common abbreviation like HTN, DM, or IHD. Try not to orally present them as most of them are non universal abbreviations. Instead present these data as hypertension, diabetes mellitus, or ischemic heart diseases.
12. With practice you will develop the capability of avoiding written presentation as this manner lack the clinical experience and make some examiner unsatisfied. The best way is to ordinarily asking the patient, writing the information, repeating them with your self, reiterating them to the patient to confirm or to correct, and finally presenting the case orally to the examiners without the case sheath.
13. Try to make a history containing even minor events. This is especially applied for undergraduate students as they may have not the capability to separate which is important and which is trivial. Once the story of the patient is complete, try to ask about the following:-
    - Admission whether to the emergency unit, intensive care unit, to the ward or the patient consulted a private clinic then admission was advised.
    - Basic investigation that the patient performed e.g. routine blood sampling, general urine examination, general stool examination, ultrasonography, chest X-ray or ECG. Each case should of course individualize.
    - Medications that the patient received.
    - Improvement or worsening in the patient's condition now. Here, you must ask about type and time of that symptom improved or worsened.
14. Postgraduate students may need in addition to recollect the history into a short medical story containing the medically correct terms. For example a 12-year-old male patient presented with a three days history of dyspnoea, chest pain and fever that then progressively worsened till the time of his admission as a confused case.

REVIEW OF OTHER SYSTEMS

This part of the history includes leadingly directed questions for each system not involved in the history of present illness. In general the body systems include:

1 Nervous system

Loss of consciousness, muscle weakness, paresthesia, headache, dizziness, convulsion, tremor, changes of smell, vision or hearing and changes of behavior or psyche

2 Cardiovascular system

Dyspnoea, PND (paroxysmal nocturnal dyspnea), orthopnea, platypnea, palpitation, chest pain, ankle edema and cyanosis

3 Respiratory system

Cough, sputum, hemoptysis, hoarseness of voice, wheezing, chest pain, dyspnoea and cyanosis

4 Gastrointestinal tract

Increase or decrease in appetite, nausea, vomiting, heartburn, haematemesis, abdominal pain, diarrhea, constipation, and rectal bleeding

5 Urological system

Loin pain, frequency, urgency, dysuria, change in the color or odor of urine, hematuria and oedema of the ankles, hands or face

6 Musculoskeletal system

Muscle ache, joint pain, swelling of joints, and limitation of joint movement

7 Integumentary system

Change in the colour of skin, skin rash, and skin itching

BIRTH HISTORY

It is desirable to divide birth history into three parts. Each of which carries particular importance in the child care and for care of the child:

1 Prenatal history

Maternal health before and during her pregnancy
Specific illness related to or complicated by pregnancy e.g., diabetes mellitus or hypertension
Any infectious diseases during pregnancy i.e. TORSCH
Radiographs taken during pregnancy
Antenatal care

2 Natal history

Gestational age of the baby e.g., preterm, term or post term
Type of labor e.g. normal vaginal delivery, caesarean section or assisted delivery
Site of labor e.g. at home or at hospital
Any complications encountered

3 Postnatal history

Onset of respiration (immediately cry)
Resuscitations efforts
Cyanosis, jaundice and convulsion
Congenital anomalies
Infection
First feeding and first bowel motion

PAST MEDICAL HISTORY

This part of history is specialized for medical problems that are neither related to the current illness nor belonging to it. For example a patient who is suffering from melena as he has duodenal ulcer so duodenal ulcer is not a past medical history, but the same patient may have epilepsy that is unrelated to melena so epilepsy is a past medical history. In general inquiring should be directed towards chicken pox, mumps, measles, rubella, whooping cough, heart failure, chronic renal disease, diabetes mellitus, and asthma.

PAST SURGICAL HISTORY

The past surgical history should include all important operations from infancy onward that are unrelated to or belonging to the presenting illness.

It is noteworthy to record circumcision in males and tooth extraction in both males and females. It is wise to ask about the operation in the form of cause of operation, type of operation, time of operation, and the post-operative complications. Ritual circumcision is practiced on Muslim and Jewish infants. The parents should be advised to perform circumcision to their child by a surgeon. This procedure if not properly performed it may result in amputation of the distal part of the glans, sepsis, removal of an excessive amount of foreskin and occurrence of urethrocutaneous fistula. Advantages of circumcision includes penile carcinoma becomes exceedingly rare, prevents phimosis and paraphimosis, decreases incidence of urinary tract infections.

VACCINATIONS

The schedule for immunization is that recommended for routine protection of children. Immunization is defined as the process of inducing immunity artificially by administration of antigenic substance. It is generally divided into two types:

1. Active immunization which includes the administration of vaccine or toxoid that stimulate the immune system to produce antibodies and cellular immunity response
2. Passive immunization which includes administration of exogenously processed antibodies. This type provides only a transient protection

Immunization schedule includes the following:

First week of life	BCG, OPV_0 and HBV_1
Ask about BCG as follow:	Injection in the left upper limb. Inspect the area then decide; BCG with scar BCG with out scar
Ask about OPV_0 as follow:	Two or three drops taken orally Did the baby vomit? If so, does the vaccination repeat?
Ask about HBV1 as follow:	An injection in the thigh or gluteal region
2 months completed	DPT_1, OPV_1, HBV_2
4 months completed	DPT_2 , OPV_2
6 months completed	DPT_3, OPV_3, HBV_3
9 months completed	Measles vaccine
15 months completed	MMR
8 month completed or one year after third primary dose	Booster one of DPT and OPV
Fourth to sixth years age	Booster two DPT and OPV
7 years age	OPV, MMR, Td
Sixth primary school	Rubella
First secondary school	OPV, MMR, Td

A child not vaccinated before one year needs the following schedule:

First visit	DPT_1, OPV_1, HBV_1
One month later	MMR
One to two months later	DPT_2, OPV_2, HBV_2
One to two months later	DPT_3, OPV_3, HBV_3
15 – 18 months later	Booster DPT, OPV
Before the age of 6 years	BCG

Contraindications and adverse reaction to vaccinations are shown in BOXES 16 – 18 and 16 – 19 respectively.

FEEDING HISTORY

Comfortable and satisfying feeding is essential for building up a healthy child. Feeding should be initiated as soon as possible to maintain normal metabolism and growth. Infants with proper feeding acquire normal mile stones, gain weight properly and develop a resistance to infection. It is, however, still possible to make mistakes by feeding too much or too little. The methods of feeding includes exclusive breast milk feeding, bottle feeding, and breast milk mixed with bottle feeding.

BOX 16 – 18. Contraindication for vaccination

Immunization should be delayed in cases of sever illness with fever

Live attenuated vaccine should not be administered to:	Because these vaccines are prepared from yolk sac, true allergy to egg is the main contraindication to:
• Immune deficient persons. • Persons with immune suppressive therapy • Persons with radiotherapy • Persons with anti-metabolite therapy	• Measles • Yellow fever • Mumps • Influenza

Typhoid vaccine is absolutely contraindicated to children younger than 2 years and to pregnancy

Discontinuation of DPT is recommended if high fever, convulsion, neurologic symptoms or shock occur

BOX 16 – 19. Adverse reactions to vaccination

Local reactions	
Immediate pain Tenderness lasts several hours or entire day	Painless nodule
Systemic reaction	
Fever GIT symptoms as diarrhoea and/or abdominal pain Urticarial rash Renal symptoms as simple proteinuria, nephrotic syndrome or hematuria Neurological symptoms	Paralytic complications Articular complications Lymphadenopathy Teratogenic complications Skeletal complications e.g. osteitis Hematologic complications e.g. thrombocytopenia

EXCLUSIVE BREAST MILK FEEDING

The aim is to increase the interval between each feed and subsequent one about 2 – 5 hours because less than 2 hour may cause inhibition of prolactin and decreases milk production. Other aim is to increase the time of each feed. Ideally, in the first 2 weeks, the baby needs seven feeds in a day; each feed lasts 20 minutes; 10 minutes in each breast. Few weeks later, the baby needs 6 feed in a day; few weeks later, the baby needs five feeds a day. Contraindications to breast feed are shown in BOX 16 – 20.

BOX 16 – 20. Contraindications for breast feeding

Absolute contraindications

Phenyl ketonuria and galactosemia

Relative maternal contraindications		Relative infantile contraindications
Septicemia Nephritis Eclampsia Profuse haemorrhage Active tuberculosis	Typhoid fever Malaria Chronic poor health Drug abuse Severe neurosis	Extremely premature baby Congenital anomalies as anencephaly Breast milk jaundice (temporary) Acute illness e.g., pneumonia (temporary)

Drugs

Contraindicated		Avoided	
Antimetabolites	Gold salts	Aspirin	Metoclopramide
Bromocriptines	Heroin	Atropine	Metronidazole
Cimetidine	Immunosuppressant	Bromides	Narcotics
Chloramphenicol	Iodides	Calciferol	Phenobarbital
Cocaine	Lithium	Estrogens	Psychotropic drugs
Cyclophosphamide	Methimazole	Ethanol	Sulfasalazine
Cyclosporine	Radioactive substances		
Diethylstilbestrol	Tetracycline		
Ergots	Thiouracil		

BOTTLE FEEDING

Ideally there should be six bottles kept in a sterile box, properly sterilized to properly prepare the milk. The ways of sterilization include:

1 Physical sterilization

Boil the feeding bottle to 10 minutes except the teat that should be removed when boiling start

2 Chemical method

Use sodium hypochloride solution 1%; add 5 ml in 1 liter of water (1 table spoonful) but this should not be kept in metallic container because it may damage it thus use a glass or plastic container. Another chemical sterilizer is sodium dichlorozocyanorate tablet; one tablet in 2 liters of water. In chemical sterilization, put the bottle 20 minutes, remove it, and then rinse it by tap water. The chemical solution is used only for 24 hours then dispose it

The milk can contains a measure spoon and the bottle is divided into volumes by lines; each spoonful milk is added to one volume. One volume is called ounce (oz) which is equal to 30 ml of fluid and equal to 20 kcal.

HOW TO CALCULATE BODY REQUIREMENTS:

Body requirement = expected slandered weight per kg x requirements (Kcal)

Expected slandered weight = Age in months + 9/2 (up to 1 year of age).

An example, a baby aged 5 months and his weight is 4 Kg. The expected weight is 5 + 9/2 = 7 Kg. The baby needs 100 Kcal/Kg/day = 100 x 7 = 700 Kcal / kg / day. Since every ounce (oz) = 20 Kcal. So 700/20=35 oz/day. In a day, give 6 meals; 35/6 = 6 oz/meal. Since 1 oz = 1 spoon. So for this baby, we give 6 spoonful of milk to 6 volumes of bottle each meal. If the baby can take more, you can reach up to 200 Kcal per Kg per day. In Kwashiorkor, the baby is ill so start with 50 Kcal per Kg per day and gradually increase it up to 100 Kcal/Kg/day. While in Marasmus, the baby is eager to feed so start normally 100 Kcal / Kg/day and increase it up to 200 /Kg/day. Kilocalorie (kcal) is the unit of heat in metabolism and used to refer to the energy content of food. Kcal is defined as the amount of heat necessary to raise the temperature of 1kg of water from 14.5 to 15.5 °C. The average caloric requirements of full term infants are about 80-120 kcal/kg during the first few months of life and about 100 kcal /kg by 1year of age.

THE WAY TO TEST THE PATENCY OT TEAT HOLE

Hold the teat up side down and look to the drops. Six drops per minute is enough. If less than 6 drops, hold the teat and push a hot needle quickly in it. If more than 6 drops per minute, change the teat. Do not squeeze the teat to test its patency.

DEVELOPMENT HISTORY

The data generated from evaluation of the developmental mile stones are greatly emphasized if the physician together with the parents is involved in its evaluation. Normal mile stones are:

At 1 month	Social smiling
At 3 months	Says Ah and Nagha
At 5 – 6 months	Turns from prone to supine
At 6 – 6.5 months	Sits with hand or pelvic support
At 7 months	Turns from supine to prone
At 8 – 9 months	Sits a lone with no support
At 9 months	Starts standing, crawling, and says Baba and Mama
At 13 months	Says few words beside Baba and Mama
At 15 months	Walking a lone Walking a lone
At 2 years	Running and can control sphincter during day time
At 3 years	Rides a tricycle and can control sphincter during night time
At 4 years	Goes to toilet a lone
At 5 years	Dress and undress a lone
At 6 years and above	Ask about school history as follows: Is he or she older in age than his/her friends? If so, why? Ask the child if he likes school!!! Does the child miss much time from the school and why? How does the child react with his/her siblings or friends of the same age?

FAMILY HISTORY

Specific questions will provide a sufficient data about the nature of the child's family:

1. Ages of the parents
2. Degree of relation between mother and father (first degree or second degree)
3. How many children are there in the family?
4. What are their ages and sexes?
5. Any stillbirths, abortion or other childhood death in the family?
6. Any family history of asthma, eczema, hay fever or any inherited illness?

SOCIOECONOMIC HISTORY

This is a valid part of history because it gives an idea about the educational level of the parents and the surrounding environment in which the child will grow. Discussion of the social elements may provide a clue to the underlying illness. Poorly educated or illiterate parents face a lot of difficulties understanding the child's need for continuity of care and appreciation of growing. Following points need to be clarified:

1 Are the parents working? What is their working?
2 Are they educated or illiterate?
3 Where do they live e.g. rural or urban area?
4 How many rooms are there in their house?
5 Water supply; tap water, well water, river water, or purified water
6 Domestic animals whether indoor or outdoor
7 Sewage system

DRUG HISTORY

History of adverse drug reaction is essential in children. The most famous adverse reaction is penicillin allergy and anaphylaxis. In addition, 10% of patients allergic to penicillin are also allergic to cephalosporin. Sulfa drugs have also known to cause allergic reactions. The most common manifestation of adverse drug reactions in children is cutaneous eruptions. Ask also whether the child is on chronic therapy e.g. steroids, chemotherapy, or anticonvulsants.

CASE PRESENTATION IN PEDIATRICS

The child name is Banen Ali Omer Alwasity who is 3 years old, male, living in Karbala/Al Amel quarter. His next of kin is (the history was taken from) his grandmother. Banen is admitted into the hospital on 13/8/2005 chiefly complaining from twitching of the face and jerking of the extremities for one hour prior to admission.

Regarding history of present illness
The condition started as a fever for 1 day prior to admission, of a sudden onset and continuous along that day, but slightly relieved when cool sponging was used. Fever was of a high grade associated with slight sweating twice or thrice at that day. The family unfortunately neglected the case. At the night of that day about 1 hour prior to admission the child started to have a generalized twitching of the extremities and face with fluttering of the eye brows that was lasting few minutes. The mother immediately splashed cold water on the child's face and whole body thus the child regained consciousness, but became tired. The family then brought the child directly to the emergency unit of the hospital where the child had been immerged in water. The child after that received injectable form of medications and syrup. The senior pediatrician advised the family to admit their child into the ward. Indeed the senior pediatrician had reassured the family that every thing is under control and admission is a daily routine to find out the cause of illness and to exclude other possibilities. Now the child feels better where the body temperature starts to decrease and the child to regain full activities.

Regarding review of other systems
Cardiovascular system. No pallor, no cyanosis, no oedema, no SOB, no palpitation or chest pain.
Respiratory system. No cough or haemoptysis.
Gastrointestinal tract. Poor appetite during the
previous day, no vomiting, no diarrhoea, no constipation or abdominal pain.
Genitourinary system. No frequency, no dysuria, no change in odor or appearance of urine or loin pain.
Musculoskeletal system. No joint pain, no muscle ache and no limitation of joint movement.
Integumentary system. No skin rash.

Regarding birth history
Prenatal history. Good maternal health before and during pregnancy. No specific illness related to, or complicated, by pregnancy. The mother experienced no skin rash or fever during her pregnancy suggestive of TORSCH infection. No x-ray had been taken during pregnancy. Poor anti-natal care as the mother did not regularly consult a primary health center whilst pregnant.
Natal history. Full term baby delivered vaginally at home with no complications encountered at that time.
Postnatal history. When Banen was newborn, he had immediately cried and needed no resuscitations efforts. He was not cyanosed not jaundiced and had no congenital anomalies or infection. His first feeding was after half an hour and his first bowel motion was at the first day of birth.

Regarding past medical history. Negative past medical history.

Regarding past surgical history. Circumcision had been adopted by the age of two years.

Regarding vaccinations history
The family unfortunately neglected the vaccinations schedule. They only adopted the vaccinations at first week of his life. He received BCG with no BCG scar, OPV_0 and HBV_1.

Regarding feeding history
The child was a breast fed up to the age of 2 yrs then the family planed a rearing process. The child now feeds the usual table foods of the family.

Regarding developmental history
The child did socially smile at the first month of age, said ah and ngha at the third month of age, turned from prone to supine at the sixth month. At the seventh month, he started to turn from supine to prone position. At the ninth month, he started to crawl and say Baba and Mama. At the 13th month, he started to say few words. He walked alone at the 15th month then run at the second year of his life. At the third year, Banen started to goes up stairs alternating feet, knows his name and rides a tricycle.

Regarding family history
The mother is 21 years old and the father is 25 years old. The father is her cousin. Their family consists of 3 children in addition to the parents; the older child is 5 years old male, the next is Banen and the younger is female aged 16 months. The family has no history of abortion or stillbirth, but both of the parents had a previous history of febrile convulsion.

Regarding social history
The mother is a house-wife and completed only her primary school study. The father had completed his intermediate school study and now is a baker. The family is living in an urban area in a rented house, supplied by tap water and proper sewage system. There is no indoor or out door domestic animals.

Regarding drug history
No history of adverse drug reactions or chronic use of medicines

CASE PRESENTATION IN PEDIATRICS

The child name is Zainb Nasir Hamza Al jubory who is 13 months old, female, living in Diwaniya/Al Hamza/Al-Asqan. Her next of kin is her mother. Zaineb is admitted into the hospital on 9/10/2004 chiefly.

Regarding chief complaint and its duration
Fever for 21 days prior to admission.

Regarding history of present illness
The condition is started as an abrupt onset low grade fever that gradually rose up to high grade, continuous all over the day; this fever was experienced together with a mild sweating especially at night. The family had used cool sponging in a way to decrease body temperature. The condition, however, soon recur after few minutes. Two days later, the family consulted a primary health centre where two types of syrups were prescribed combined with suppositories. Unfortunately, five days with this treatment had not lead to improvement in their child's suffer. The family after that revisited the primary health center. Referral to a local hospital was advised, but the family ignored the advice and started again to use a cool sponging together with the same medicines prescribed earlier.
The condition of the child deteriorated more and more. The child became reluctant to feed and started to have little amount, white colored, effortless vomiting infrequently experienced twice to thrice per day together with green colored frequent bowel motions about 8 motions per day, loose in nature, accompanied by passage of a mucus. The child in addition suffered from abdominal pain, experienced as a screaming all over the abdomen as well as non productive cough experienced mainly at night.
The family thus consulted a private clinic where admission into the children hospital was advised.
The child was directly received i.v. fluid in the form of isotonic saline and oxygen with high flow and concentration at the emergency unit. After admission to the ward, general stool examination as well as CXR had been performed together with blood investigation.
The child received thereafter I.V. fluid, and I.V. drugs as well as a blood transfusion; one unit was adopted.
The child continues to receive injectable form of medications and becomes generally better than the time of admission where the bowel motions return to the pre-illness state, vomiting is stopped, cough is subsided and body temperature is controlled.

Regarding review of other systems
Nervous system. No fit, but a sharp decrease in the activity of the child.
Cardiovascular system. Pallor was present but with no cyanosis, dyspnea, or orthopnoea.
Genitourinary system. No frequency, no dysuria, no change in odor or appearance of urine.
Musculoskeletal system. No joint pain, no muscle ache, no limitation of joint movement, but generalized inability to perform usual daily activities.
Integumentary system. No skin rash.

Regarding vaccination history
The child received BCG, OPV0 and HBV1 at first week of life with a BCG scar at the left deltoid region. At the second month of life, she received DPT1, OPV1 and HBV2 then the family neglected the vaccination schedule.

Regarding feeding history
The child is a bottle fed; improper sterilization and preparation.

Regarding developmental history
She started to smile at the 4th week of life; she can't crawl yet.

Regarding family history
The mother is 38 years old and the father is 45 years old. They are a second degree relative. The family consists of 10 members. The older one is male and aged 20 years; the next is male and aged 18 years; female and aged 16years; female aged 14 years; female aged 10 years; female aged 8 years; female aged 6years; female aged 8 years and the youngest is Zaineb. The mother had a positive family history of abortion.

Regarding social history
The father is a farmer and the mother is a house wife. Both of them are illiterate. The family is living in a rural area, in their own house that consists of 3 rooms but not supplied by tap water. The water supply is from an artificial well. They have indoor domestic animals.
No sewage system is present.

Regarding Drug history
No allergy to penicillin.

GROWTH CHARTS

Measurement of growth elements represented by growth charts is essential practice for the reliable interpretation of the growth data.

SUPINE LENGTH OR HEIGHT

Supine length can be more accurate in children under the age of 3 years. It is measured as the child lies on a firm mattress or table with the head is firmly held against the end of the mattress. A measuring tape is used to measure the length from the feet to the head (FIGURE 16 – 1). Standing height is generally less by 1 cm from that of the supine length but this should not be added to the result of the standing height. Standing height is measured as the child stands erect, with heels, buttocks, upper part of the back and occiput placed against the wall. Average gain in early school age is 6 cm/year. Normal values of length or height are shown in BOX 16 - 21.

WEIGHT

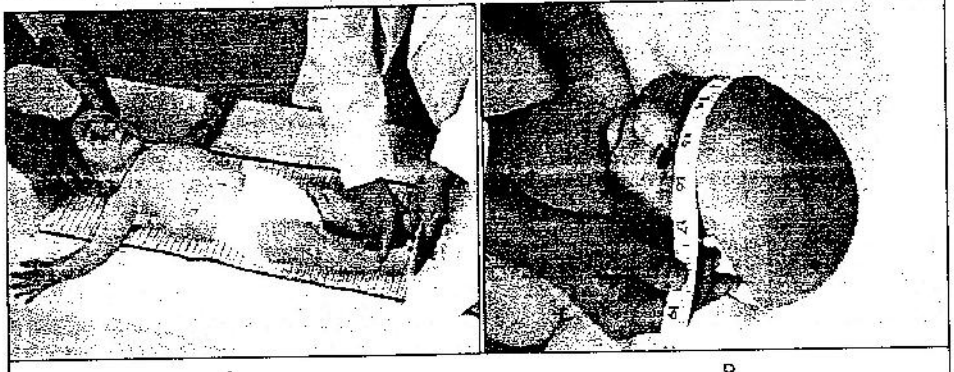
The full term infants generally double birth weight by 5 months and triple it by 1year. Average gain in weight in early school age is 3 – 3.5 kg/year. Normal values of weight with respect to the age are shown in BOX 16 - 21.

HEAD (OCCIPITOFRONTAL) CIRCUMFERENCE (OFC)

The measuring tape is applied firmly over the glabila and supraorbital ridges anteriorly and turns around the occiput to give maximal circumference (FIGURE 16 – 1). The head circumference is normally 35 cm at birth and increases to approximately 44 cm by 6 months and to 47 at the end of one year. Normal values of OFC with respect to the age are shown in BOX 16 - 21.

BOX 16 - 21. Normal growth values

Length or height	Normal values	Weight	Normal values	OFC	Normal values
At birth	50 cm	At birth	2.5 – 4.5 Kg	At birth	Each 1month x 2 cm
6 months	65 cm	UP to 1 year	(Age in months + 9)/2	3–6 months	Each 1 month x 1 cm
1 year	75 cm	1 – 6 years	(Age in years + 4) x 2 or (Age in years x 2) + 8	6 m – 1 year	Each 1 month x 1/2 cm
2 years	85 cm	6 – 12 year	(Age in years) x 3	Age of 2 years	49 cm
3 years	95 cm			Age of 12 years	54 cm (adult size)
4 years	100 cm				



A B

FIGURE 16 – 1. Measurement of the supine length (A) and head circumference (B).

INTERPRETATION OF THE RESULTS

The results are then applied over the growth charts that consist of 3rd, 10th, 25th, 50th, 75th, 90th, and 97th percentile. Then observe the following:

1. A child presenting for the first time outside the area between the 10th and 90th percentile should be regarded with slight suspicion unhealthy
2. Those outside the 3rd and 97th percentile should be regarded unhealthy until proved the other wise

The percentile means for example 5 years old child, weighing 15 Kg, at 25th percentile means 25% of those aged 5 years, their weight is 15 Kg or less.

GROWTH CHARTS

TEMPERATURE

Measurement of the temperature is recommended to be taken from the axillary region rather than oral temperature. The axillary temperature is 0.5°C lower than oral temperature thus a 0.5°C is added to the axillary temperature. In infants, the temperature is about 1°C higher than in older children. There are six methods for temperature measurement (BOX 16 – 22). In general:

For children ≥ 5 years, use the oral method.

For children < 5 years, use axillary method and add 0.5° to the result.

BOX 16 – 22. Methods for temperature measurement

Method	Time	The result	Method	Time	The result
Oral	1 minute	The same	Thermal tape	2–3 minutes	The same
Rectal	1 minute	Subtract 0.5°C	Groin	2–3 minutes	Add 0.5°C
Axillary	2–3 minutes	Add 0.5°C	External auditory	2–3 minutes	The same

HEART RATE

Listen for the heart rate and rhythm only by auscultation against precordium at the mitral area. Normal heart rate average values are shown in BOX 16 – 23. A pulse rate that is too rapid to count (usually >180–200/min) usually indicates paroxysmal supraventricular tachycardia. Bradycardia in infants or young children may be due to drug ingestion, hypoxia, intracranial or neurologic conditions, cardiac arrhythmia such as heart block.

RESPIRATORY RATE

The respiratory rate in infants and children has a greater range, as that of heart rate, and is more responsive to illness, exercise, and emotion. Normal respiratory rate values are shown in BOX 16 – 23. Extremely rapid and shallow respiratory rate are seen in newborns with cyanotic cardiac disease who have normal lungs but right-to-left shunting, and in conditions of metabolic acidosis. Children with respiratory diseases such as bronchiolitis or pneumonia have rapid respirations (up to 80–90/min), but also increased work of breathing such as grunting, nasal flaring, or use of accessory muscles. The best single physical finding for ruling out pneumonia is an absence of tachypnea. Fever can raise respiratory rates in infants. In the absence of pneumonia, an infant's respiratory rate can increase by up to 10 respirations per minute for each degree centigrade of fever.

BOX 16 – 23. Normal vital signs values

Heart rate	Normal value	Respiratory rate	Normal value	Blood pressure	Normal value
Newborn	140 bpm	Since birth – 2 months	40 – 60 breaths per minute. Tachypnea > 60	In newborn	65/45 mmHg
1 year	110 bpm	2 months – 1 year	30 – 50 breaths per minute. Tachypnea > 50	At 1year	75/50 mmHg
3 years	100 bpm			At 4years	85/60 mmHg
8 years	90 bpm	1 year – 5 years	20 – 40 breaths per minute. Tachypnea > 40	At 8years	95/65 mmHg
11 years	80 bpm			At 10 years	100/70 mmHg

BLOOD PRESSURE

Measurement of blood pressure is only needed when renal or cardiovascular disease is suspected. Two methods for blood pressure measurement:

1 Sphygmomanometer

The inflatable bag should be long enough to encircle the full circumference of the upper arm with a width roughly equal to 1/3 of the length of the upper arm, forearm as far as the wrist

2 Flush method

The arm is tightly bandaged to exclude the blood up to the level of the cuff which is then inflated. The bandage is then removed to reveal a white limb. The pressure in the cuff is slowly reduced; point at which the skin flushes is an approximate indication of the systolic blood pressure

The normal values of blood pressure in the arm are shown in BOX 16 – 23. Children have elevated blood pressure during exercise, crying, and anxiety. The most frequent cause of a raised blood pressure in children (BOX 16 – 24) is probably an improperly performed examination, often due to an incorrect cuff size.

In children, as in adults, blood pressure readings from the thigh are approximately 10 mm Hg higher than those from the upper arm. If they are the same or lower, coarctation of the aorta should be suspected.

BOX 16 – 24. Causes of hypertension in pediatric patients

Newborn	Middle Childhood	Infancy and early childhood
Renal artery disease (stenosis, thrombosis) Congenital renal malformations Coarctation of the aorta	Renal parenchymal or arterial disease Primary hypertension Coarctation of the aorta	Renal parenchymal or artery disease Coarctation of the aorta

GENERAL EXAMINATION

The general examination is conducted through, as we have learned, two steps. The first step is the general look to the patient and the second step is regional examination. As you have learned earlier, a mnemonic (A SAD CBD) is used to describe the points of general look to the patient. These points are **A**ge group, **S**ex (always female), **A**wareness, **D**ecubitus (position of the patient in the bed), state of **C**omfortability, **B**ody built (BOX 16 – 25), **D**yspnoea and **D**ehydration.

BOX 16 – 25. Certain relationship of expected body weight

Normal body weight	90 – 120 % of expected weight
Obesity	> 120 % of expected weight
Kwashiorkor	60 – 80 % of expected weight with oedema
Under weigh	60 – 80 % of expected weight without oedema
Marasmus	< 60 % of expected weight without oedema
Marasmus – Kwashiorkor	< 60 % of expected weight with edema

If the baby looks dehydrated, try to determine the degree of dehydration. The degree of dehydration can be noted by applying certain parameters shown in BOX 16 – 26.

BOX 16 – 26. The degree of dehydration

General appearance		Anterior fontanel	
Mild	Alert, thirsty and restless	Mild	Normal
Moderate	Thirsty, lethargic and drowsy	Moderate	Depressed (sunken)
Severe	Drowsy, sweaty, cyanosis, and may be comatose	Severe	Very depressed (very sunken)
The eye		The tear	
Mild	Normal	Mild	Present
Moderate	Sunken	Moderate	Decrease in tearing
Severe	Very sunken	Severe	Absent tearing
The mucus membrane, especially tongue		The respiratory rate	
Mild	Moist	Mild	Normal
Moderate	Dry	Moderate	Rapid
Severe	Very dry	Severe	Rapid and deep
Skin retraction		Radial pulse	
Mild	Retract rapidly	Mild	Normal rate and strength
Moderate	Retract over 2 seconds	Moderate	Rapid and weak
Severe	Retract very slowly	Severe	Feeble and impalpable
Systolic blood pressure		Capillary refilling	
Mild	Normal	Mild	Normal
Moderate	Normal to low	Moderate	Refill in about 2 seconds
Severe	Low unrecorded	Severe	More than 3 seconds
Urinary flow			
Mild	Normal		
Moderate	Reduced		
Severe	Anuria		

In the regional examination, the approach here starts with the examination of the head and proceeds to the neck, chest, abdomen, and extremities. We used to present any case taking particular consideration to Jaundice, Anemia (look for anemia in the conjunctivae and sublingual tissues not in the palms as pregnancy is associated with palmar erythema), Cyanosis, Clubbing, Oedema, and Lymphadenopathy. These signs are collectively known as JACCOL.

THE HEAD

While the baby lies on the mother's lap, examine the head for any odd appearance. The head is examined topographically in the following steps:

SHAPE AND SIZE

Shape and size of the skull should be documented carefully. Hydroccphaly is a large cranial vault due to enlarged ventricles. Microcephly is a small cranial vault. Oxycephaly or turricephaly is enlarged skull with prominent vertex. In Brachycephaly, the anteroposterior diameter is greatly reduced and the occiput flattened. It may occur in mongolism. Scaphocephaly is a long narrow skull. Plagiocephaly a symmetrical skull results from fetal posture. In dolichocephaly, the anteroposterior diameter is long which is opposite to brachycephaly. Try to look to the parent's head where such shapes may be a familial trait.

SKULL

For clinical purposes, the skull is further divided into fontanelles, sutures and bones. There are six fontanelles; anterior and posterior, two sphenoidal and two mastoidal. Anterior fontanel is closed at 10-14 months, but may be closed as early as 3 months or as late as 18 months. Its size is about 2 X 2 cm in healthy child. It is pulsatile and in the same plane as the rest of the surrounding skull. Depressed anterior fontanel may be due to dehydration and became tense and bulged in case of increased intracranial pressure, meningitis and during crying. Delay in time of closure may suggest hypothyroidism, rickets, hydrocephalus, achondroplasia, Down syndrome, vitamin D deficiency, and osteogenesis imperfecta. Posterior fontanel is closed at 2 – 3 months of life. It is located by passing a finger along the sagittal suture to the junction with the lambdoidal suture. It allows admission of the tip of little finger.

There are three sutures; coronal, sagittal, and lambdoidal. These sutures tend to be separated in the neonatal period and ossified rapidly at 6 months of age. If the suture is still separated after this period, it suggests increased intracranial pressure. If the suture is prematurely fused, it suggests a condition known as craniosynostosis.

Prominence of maxillary bone may occur in thalassemia major. Bossing of frontal and parietal bones occur in rickets. Craniotabes which is a small non ossified area in the membranous bones of the skull with a crack feeling when compressed occurs in rickets (BOX 16 – 27).

BOX 16 – 27. Clinical features of rickets

Early signs	Advanced signs
Craniotabes is detected by firm pressure over posterior parietal bones and occiput. Rachitic rosary is palpable enlargement of the costochondral junctions. Palpable thickening of wrists and ankles. Increased sweating around the head.	Permanent asymmetry of the head Delayed closure of the anterior fontanel Frontal and parietal bossing Caput quadratum (box-like head) Visible rachitic rosary Pigeon chest Harrison groove which is a horizontal depression corresponds with the costal insertions of the diaphragm Scoliosis, kyphosis (in sitting), and lordosis (in erect posture) Knock-knees Rachitic dwarfism Tetany

Percussion of the parietal bone on each side of the skull using your index or middle finger will produce a cracked pot sound in normal infants prior to closure of the cranial sutures. This is called Macewen's sign. It is noted in older infants with high intracranial pressure that causes closed cranial sutures to separate as in lead encephalopathy, brain tumor, or hydrocephalus. Bruits many be heard by auscultation over an arteriovenous malformation.

THE FACE

Earthy pigmented face may suggest uremia. Congested face may suggest polycythemia. Red raised eruptions on the bridges of the nose extending to the cheek commonly in female due to systemic lupus erythematosus (SLE). This is called batwing or butterfly rash. Pale cheeks may occur in anemia or hypopituitarism or as a normal trait. Pale and puffy cheeks occur in nephrotic syndrome. Malar flush which is a bright circumscribed flush occurs due to mitral stenosis. Bluish tint of the cheeks may suggest congestive heart failure. Tender bilateral swellings of the parotid glands may suggest mumps. Unilateral swelling and reddening of the skin may suggest acute parotitis. Persistent enlargement of the parotid glands accompanied by dry tearless eyes suggests Sjögren's syndrome. Unilateral irregular and painless swelling may suggest mixed parotid tumour.

THE EARS

Check for congenital anomalies e.g., low set ears. Check then for patency of external auditory meatus.

THE EYES

Blindness may be a manifestation of congenital cataract, congenital optic atrophy, congenital cerebral agenesis or retinoblastoma. Yellowish discoloration of the sclerae occurs in jaundice; pale conjunctiva in case of anaemia. Dropping of the upper eye lid is a manifestation of Horner's syndrome or third cranial nerve palsy. A slight degree of internal strabismus is common and frequently disappears, but paralytic strabismus is often persistent. Signs of conjunctivitis should also noted. Periorbital oedema suggests nephritis or nephrosis. Blue sclerae occurs in iron deficiency anemia (IDA), osteogenesis imperfecta, Ehler Danlos syndrome, and other conditions. A child may have abnormal shape or length of palpebral fissures e.g., up-slanting (Down syndrome), down-slanting (Noonan's syndrome), and short (fetal alcohol effects).

THE NOSE

Depressed nose may be due to hypothyroidism. Nasal discharge is usually a sign of upper respiratory tract infection while retracted alae nasi suggests lower respiratory tract infection. Pale mucosa of the nose may be due to an allergic process. Check the septum for deviation, presence of a polyp or any other abnormalities.

THE MOUTH

Crack at the angle of the mouth is called angular stomatitis or cheilosis and suggest iron or vitamin B deficiency. Small blisters or crusts may suggest herpes simplex infection. Huge protruded tongue occurs in cretinism and in Down syndrome. A swollen lips may suggest allergic process while fissuring and/or ulceration suggests infection e.g., Candida (oral thrush). Blue colored lips occur in cyanotic congenital heart disease while pallor occurs in anemia. Red smooth painful tongue occurs due to vitamin B_{12} deficiency (BOX 16 – 28).

BOX 16 – 28. Effects of vitamins deficiency

Vitamin	Effects of deficiency	Vitamin	Effects of deficiency
Vitamin A	Night blindness, xerosis conjunctivae, xerosis cornea, and keratomalacia	Vitamin B1 (Thiamine)	Beriberi Wernicke's encephalopathy
Vitamin B2 (Riboflavin)	Cheilosis, glossitis, keratitis, and photophobia	Vitamin B3 (Niacin)	Pellagra; dermatitis, diarrhoea, dementia, and death
Vitamin B6 (Pyridoxine)	Convulsions, peripheral neuropathy, and dermatitis.	Vitamin B12 (Cobalamin)	Juvenile pernicious anaemia
Biotin	Seborrheic dermatitis and hallucinations	Vitamin C (Ascorbic acid)	Scurvy and poor wound healing
Vitamin D	Rickets, tetany, poor growth, and osteomalacia	Vitamin E	Hemolysis in premature infants
Vitamin K	Hemorrhagic manifestation		

In scurvy (due to vitamin C deficiency), the gums may appear swollen red with bleeding. Cleft lip and palate may be also noticeable. Adherent curds at the buccal mucosa and tongue suggest oral thrush. Dryness of the mucosa may suggest dehydration; in hypernatremia dehydration, the tongue is woody and dry. Koplik's spots are tiny grayish lesion surrounded by erythema; they are visible on the buccal mucosa due to measles. Koplik's spots are usually opposite the premolar teeth. The throat is best inspected when the child is sitting on the mother's lap, facing you. The mother should steady the child's forehead with one hand and enclose the child's arms a cross chest with the other hand. Generally speaking, signs of malnutrition are shown in BOX 16 – 29.

BOX 16 – 29. Signs of malnutrition

Hairs are brittle or lost	Nail becomes concave and brittle
Eyes	Mouth
Blue sclera (IDA) Pale conjunctiva (anemia) Long lashes (vitamin A deficiency) Pitot's spots (vitamin B deficiency)	Angular stomatitis (iron of vitamin B deficiency) Red, smooth, beefy, and painful tongue (vitamin B12 deficiency)
Skin	Chest
Skin becomes dry and shiny, petechial rash and other rashes as well as edema	Rosary due to vitamin C deficiency called scorbutic rosary

THE TEEETH

The first deciduous tooth is the mandibular central incisor. The deciduous teeth are 20 teeth and erupt in the sequence shown in BOX 16 - 30.

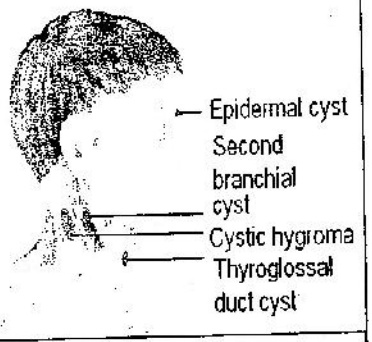
BOX 16 - 30. The sequence of teeth eruption

Central incisors		First molars	
Mandibular	5-8 months	Mandibular and maxillary	11-18 months
Maxillary	6-10 months	Cuspids (canines)	
Lateral incisors		Mandibular and maxillary	16-20 months
Mandibular	7-10 months	Second molars	
Maxillary	8-12 months	Mandibular and maxillary	20-30 months

The first permanent tooth is the first molar and erupts at the 6 years of age. The permanent teeth are 20 teeth. The last tooth is erupted at the 16 – 21 years of age. It is therefore called the wisdom tooth which is third molar tooth.

THE NECK

Beyond infancy, the techniques for examining the neck are the same as for adults. Detailed examination of theses elements are described earlier. Because the necks of infants are short, it is best to palpate the neck while infants are lying supine, whereas older children are best examined while sitting. Malignancy is more likely if the node is greater than 2 cm, is hard or fixed to the skin or underlying tissues i.e., not mobile, and is accompanied by serious systemic signs such as weight loss. Thyroglossal cysts are located at the midline of the neck, just above the thyroid cartilage. These are small, firm, mobile masses that move upward with tongue protrusion or with swallowing. They are often detected after two years. Branchial cleft cysts appear as small dimples or openings anterior to the mid portion of the sternocleidomastoid muscle (FIGURE 16 – 2). They may be associated with a sinus tract. Preauricular cysts or sinuses are common, pinhole size pits, usually located anterior to the helix of the ear. They are often bilateral and may occasionally be associated with hearing deficits.



FIGUER 16 – 2. Some neck masses.

THE CHEST

Much can be learned by examination of the chest. It includes examination of the respiratory system and examination of the precordium (discussed later).

THE EXTREMITIES

External features of the hands are of great importance and attention should be paid to any minor changes or Congenital anomalies. Syndactyly is the most common hand congenital anomaly. It means a fusion or webbing of two digits. Extradactyly (polydactyly) is the second common hand congenital anomaly. It means presence of more than 5 digits. The signs and symptoms of foot pathology are generally pain, swelling, deformity and sensory changes. The foot is normally in plantigrade position. If not in this condition, it is called talipes (club-foot). The deformities are named according to the presenting position. These are the equines (flexion), calcanus (extension), varus (inversion), and valgus (eversion). There is also a combination of two types of deformities e.g., equinovarus (flexion and inversion) or calcaneovalgus (extension and eversion). To the above mentioned deformities, we can add cavus which means hollowing of the instep. The commonest deformity of these is the talipes equinovarus. The causes of such a deformity are genetic cause, trauma, drugs, and some mal-position in uterus. Pes cavus is characterized by hollowing of the instep. The congenital form is due to shortening of the palmer fascia and the acquired form is due to paralysis of the small muscles of the foot. In the feet, there are two arches; longitudinal and transverse arches. Flattening of the longitudinal arch will result in pes planus (flat foot) which is one of the most common defects of the feet while flattening of the transverse arch will result in transverse flat-foot (splay foot). Edema of the leg should also be approached as described in the earlier chapters.

SYSTEMIC EXAMINATION

The same procedures and techniques used for adult patients are applied to pediatric patients although some modifications are needed sometimes.

THE RESPIRATORY SYSTEM

INSPECTION

Look at the symmetry of the chest, shape of the chest, expansion of the chest, pattern of breathing, dyspnea, and texture of the chest. Listen then for heard sounds and count the respiratory rate. The techniques needed are the same as that used for adults. Obstructive respiratory disease in infants can result in Hoover's sign, or paradoxical see-saw breathing, in which the abdomen moves outward while the chest moves inward during inspiration. This thoracoabdominal paradox, inward movement of the chest and outward movement of the abdomen during inspiration, is a normal finding in preterm and newborn infants. It continues to persist during active sleep even when it is no longer seen during wakefulness or quiet sleep because of the decreased muscle tone of active sleep. As muscle strength increases and chest wall compliance decreases with age and growth, this paradox is no longer discernable as a normal finding. Grunting sound can be heard during listening to the chest and is produced by forceful expiration against partially closed glottis due to hypoxia, pulmonary edema and pneumonia. Harrison's sulcus (FIGURE 16 – 3) is an anterolateral depression of the thorax at the insertion of diaphragm due to asthma and nasopharyngeal obstruction. Most of the child's breathing is diaphragmatic and produce quite marked abdominal movement and a little chest movement.

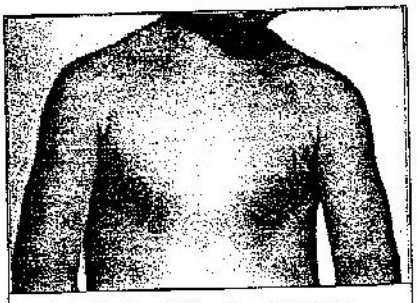


FIGURE 16 – 3. Harrison's sulcus which is bilateral depression of the rib cage in the infra-mammary regions.

PALPATION

Feel foe chest tenderness and search for thickening at the costochondral junction (rickety rosary) that is due to rickets. Localize then the apex beat, examine both axillae for lymph nodes, and ascertain the location of the trachea. In children < 5 years of age, the apex beat is at the fourth intercostal space just to the left of the midclavicular line. In children >5years, apex beat is at the fifth intercostal space in the midclavicular line. Lastly, determine his/her chest expansion and vocal fremitus if the child is reasonable age and cooperative. The techniques needed and the clinical findings are the same as that of adults.

PRCUSSION

Percussion is useful in older children and is generally conducted over the chest up to the tenth rib as the liver and kidneys can give rise to dull percussion notes. The percussion notes are resonant over all the chest regions apart from liver and cardiac dullness. The techniques needed and the findings are the same as that of adults.

AUSCULTATION

If you ask young children to take deep breaths, they will often hold their breath, making it even more difficult for you to auscultate over the lungs. Thus it is easier, for preschool children, to let them breathe normally. For older children, you can demonstrate how to take nice, quiet, deep breaths, and make a game out of it. Generally, listen to the air entry over both lungs, determine the type of breath sounds and any added sounds (wheeze, crackles, etc.), and lastly perform vocal resonance if the child is in reasonable age. In the presence of upper airway obstruction such as croup, inspiration is prolonged and accompanied by other signs such as stridor, cough, or wheeze. In the presence of lower airway obstruction such as asthma, expiration is prolonged and often accompanied by audible wheezing. The techniques needed and the findings are the same as that of adults.

PERCORDIAL EXAMINATION

INSPECTION

Look at the shape of the chest, presence of scars, and any visible pulsation. The techniques needed and the findings are the same as that of adults.

PALPATION

Localize the apex beat and determine its character. Feel then for heaves (lifts), thrills, and palpable heart sounds. The techniques needed and the findings are the same as that of adults.

PERCUSSION

Percussion seldom carried out like in adults. The percussion note over the chest becomes hyper-resonant when there is lung hyperinflation which masks the dullness over the heart and liver.

AUSCULTATION

Auscultate all the precordial areas (mitral, left parasternal, Erb's, pulmonary, and aortic areas) taking particular consideration to the Erb's area where VSD murmur is maximally heard. Listen the first heart sound, second heard sound, and any added sounds. Heart rate should be counted in addition. First heart sound (S1) is single; second heart sound (S2) is generally splitting and is best heard at the second intercostal space; third heart sound (S3) may be heard at the apex of the heart. The techniques necessary for cardiac murmur (BOX 16 – 31) are the same as that for adults.

BOX 16 – 31. Timing of cardiac murmurs

Systolic murmurs	Diastolic murmurs	Continuous murmurs
Ejection systolic murmurs • Aortic stenosis • Pulmonary stenosis • Atrial septal defect (common)	Early diastolic murmurs • Aortic regurgitation • Pulmonary regurgitation	Patent ductus arteriosus Venous hum Coarctation of aorta Arteriovenous fistula (coronary artery, pulmonary, systemic) Aortopulmonary connection (e.g. congenital, Blalock shunt) Rupture of sinus of Valsalva into right ventricle or atrium
Pansystolic murmurs • Mitral regurgitation • Tricuspid regurgitation • Ventricular septal defect (common)	Mid-diastolic murmurs • Mitral stenosis • Tricuspid stenosis	
Late systolic murmurs • HOCM • Mitral valve prolapse	Austin flint murmur	

ASD murmur produces an ejection systolic murmur. The area of maximum intensity is the second left intercostal space (pulmonary area). This murmur is harsh in quality, radiating into left shoulder and of any grade. VSD murmur produces a pansystolic murmur. The area of maximum intensity is left parasternal area. This murmur is of rough quality, propagated into the axilla and of any grade. PDA murmur produces continuous murmur. Its area of maximum intensity is second left intercostal space, machinery in quality (to and fro). It is due to a link between aorta and the main pulmonary artery at a point close to the origin of left subclavian artery. The baby has collapsing pulse and differential cyanosis.

ABDOMEN EXAMINATION

INSPECTION

Look at the pattern of movement, contour (shape) of the abdomen, and the texture of the abdomen. The techniques needed and the findings are the same as that of adults. When the baby cries note any hernial orifice. Generalised abdominal (symmetrical) distension may be due to fat, flatus, feces, fluid, functional (FIVE Fs).

PALPATION

Palpate the abdomen superficially for superficial masses and tenderness and deeply for deep masses and tenderness as well as for enlargement of abdominal viscera. The normal liver span for these elder than 6 years is more than 6 cm.

PERCUSSION

Percuss the abdomen to determine liver span, bladder (suprapubic percussion), and to ascertain the presence of ascites (shifting dullness and fluid or transmitting thrill). The techniques needed and the findings are the same as that of adults.

AUSCULTATION

Listen at Mc Burney's point for bowel sounds and at epigastrium for succession splash. Listen also for arterial bruits and for venous hum.

GENITALIA

For male genitalia, note the penis for any congenital anomalies e.g. hypospadius; note the scrotum for any atrophy or absence of creases which may suggest undescended testis. If so; make sure that your hands are warm and place a finger on the line of inguinal canal and advance it towards the scrotum to prevent cremasteric reflex then palpate it at the scrotum. If the testis is undescended, you will fell it in the inguinal canal, but if you can not feel it so it is either abdominal undescended testis or ectopic testis. The differential diagnosis for undescended testis is retractile or migratory testis that can be pulled down into scrotum and descend when the child sit and cross his legs.

For female genitalia, check the vulva for soreness or discharge; check for fusion of labia; check for enlargement of clitoris (clitoromegaly).

NEUROLOGICAL EXAMINATION

HIGHER CEREBRAL FUNCTION

Determine the level of the child's consciousness (drowsy, lethargic, obtunded, stuporous, or comatose). The speech and other cognitive functions are tested later in childhood. The techniques needed and the findings are the same as that of adults.

CRANIAL NERVES EXAMINATION

The techniques needed to test the cranial nerves will be modified according to the age of the child:

FIRST CRANIAL (OLFACTORY) NERVE

Specific appreciation of smell does not develop until later in childhood. At that time use the techniques described under the heading of nervous system examination.

SECOND CRANIAL (OPTIC) NERVE

Vision is present since birth. It is usually tested in the following ways:

At 4 weeks	The infant will watch the mother
At 6 weeks	The infant follows moving object, but arc of vision is < 90°
At 8 weeks	The infants can fix, converge and locus on an object
At 12 weeks	The arc of vision is more or equal to 180°

Thus at 6 – 8 weeks you can detect a defect in visual perception and 12 weeks you can detect a defect in both eye movement

THIRD (OCULOMOTOR), FOURTH (TROCHLEAR), AND SIXTH (ABDUCENS) CRANIAL NERVES

These cranial nerves are examined together. Remember that the steps of examination (3 x 3) include prime position for ptosis, proptosis, and inequality of pupil, ocular movement for nystagmus, limitation of eye movement, and squint and lastly pupillary reaction for direct light reflex, indirect light reflex, and accommodation reflex. In infancy, horizontal and vertical movement can be assessed by spinning the infant through 360° of arc; this is for horizontal plane, but vertical plane can be tested by tilting the vertically held infant into a semi-prone position. In late infancy, the infant is able to follow attractive objects e.g., toys in vertical and horizontal directions. Squint is a common problem and a transient in early weeks of life, but if it persists more than this period it will be significant and could be paralytic and non-paralytic. In paralytic squint, the eye fails to move in one or more directions. In non-paralytic squint eye, the squint is more obvious at the end of the day i.e. when baby became tired and squint eye can be tested when the child looks at a far object and cover the other eye. Thus the squint eye will deviate to look at the object, but the gaze will be more away from the object. When you cover the squint eye, the dominant eye will fix to a far object and remain fixed.

FIFTH CRANIAL (TRIGEMINAL) NERVE

For motor components, inspect temporalis and masseter muscles then try to palpate these muscles. For reflexes, test corneal and jaw reflexes. For sensory components, test sensation at ophthalmic, maxillary and mandibular areas.

SEVENTH CRANIAL (FACIAL) NERVE

The child can be made to smile or even when cry to inspect any a symmetry of facial movement. In lower motor neuron lesion, both upper and lower halves of the face will be affected. In upper motor neuron, only the lower half of the face will be affected.

EIGHT CRANIAL (VESTIBULOCOCHLEAR) NERVE

This cranial nerve is tested as follows:

At 6 weeks	Startle test is used as follows; when you make a sudden loud sound, the baby will startle or even cry
At 6 months	Hearing is tested by a sound made at 45 cm away from the ear at the level of the ear but slightly behind using a rattle
At 10 months	The same test as the above is used, but the distance is 90 cm and the level is above or below out of infant's sight

NINTH CRANIAL (GLOSSOPHARYNGEAL) AND TENTH CRANIAL (VAGUS) NERVES

These are usually examined together for gag reflex and palatal reflex. If the child has inspiratory strider, you may suggest a vagus nerve lesion due to recurrent laryngeal nerve involvement.

ELEVENTH CRANIAL (ACCESSORY) NERVE

The strength of trapezius and sternomastoid muscles is tested.

TWELFTH CRANIAL (HYPOGLOSSAL) NERVE
Many young infants protrude their tongue out thus inspect it for tremor, wasting and deviation.

MENINGEAL SIGNS
Perform neck stiffness test, Kernig's sign, and Brudzinski's sign.

MOTOR SYSTEM EXAMINATION
Motor system examination includes inspection and palpation of the muscles, tone assessment, assessment of the power of the muscles, assessment of the reflexes, and cerebellar signs.

INSPECTION AND PALPATION
Look at the child's posture, inspect and palpate the muscles, and lastly look for any abnormal movements including tapping the inner thigh and shoulder to elicit fasciculations.

TONE ASSESSMENT
Assess the tone via the following steps:

1. Flaccid posture indicates hypotonia
2. Passive movement of joints as adults
3. Head lag; pick up the child from beneath back there will be head lag in hypotonia
4. Pick up the child from arm pits, he/she will impress slipping through your hands in case of hypotonia
5. Ventral suspension; pick up the child from beneath abdomen, a U-shape posture will be assumed in hypotonia
6. Gower's sign; ask the child to climb steps or stand up from a prone position, weakness in the proximal lower limb muscles causes the child to use the hands to climb up the legs in order to assume an upright position

POWER (STRENGTH) OF THE MUSCLES
The power can be assessed by watching physical activities of the child or by looking at the child while he/she picks up small objects.

REFLEXES
Assess the superficial and deep reflexes as we had learned earlier. The superficial reflexes include the abdominal reflex (T8–T12), cremasteric (L1–L2), and planter flexion reflex (S1–S2). Planter response is extensor up to18 months but if persists beyond this time it indicates an upper motor neurone lesion. Deep tendon reflexes include the triceps reflex (C6–C7), biceps reflex (C5-C6), supinator reflex (C5–C6), knee reflex (L3–L4), and lastly ankle jerk (S1). Upper motor neurone (UMN) lesion causes brisk reflexes and lower motor neurone lesions (LMN) lesion causes hyporeflexia or areflexia.

CEREBELLAR COORDINATION
In older children, you can perform nose finger test for intention tremor and past pointing, dyssynergia, rebound phenomenon, dysdiadochokinesis and heel shin test. You can modify nose-finger test using a toy held in the hand. If the child is old enough, watching him/her dress or do up shoelace which is a good method. In addition to nystagmus, ataxia and scanning speech can be noticed.

SENSORY SYSTEM EXAMINATION
Testing of sensation in young children is difficult and probably best omitted unless there is a strong suspicion of neurologic deficit. If it is indicated, like that in adults, test for:

1. Superficial sensation

Test light touch, pain sensation, and temperature

2. Deep sensation

Test position and vibration sense

3. Cortical function

Over the age of 5 years, cortical functions testing are possible

MUSCULOSKELETAL SYSTEM EXAMINATION

The techniques needed are the same as that of adults and include three steps; look, feel, and move. Look at scar that may indicate previous operations and skin colour changes e.g. blue (cyanosis), red (inflammation). Look also at the creases. Abnormal creases indicate deformity; shiny skin with no creases indicates a swelling. Look for muscle wasting and swelling (lump). Feel the temperature, pulses, and assess any swelling. Look for joint tenderness. Actively (what the patient did) and then passively (what you did) move the joints. At certain occasions you may need to perform special tests.

SCREENING MUSCULOSKELETAL EAXMINATION (FIGURE 16 – 4)
If he/she is in reasonable age, ask the child to perform the following actions:

1 Stand straight, facing you
2 Move neck in all directions; look at the ceiling and floor, touch ears to shoulders
3 Shrug shoulders while you hold them down
4 Hold arms out to side and lift arms while you press down
5 Hold arms out to side with elbows bent 90°, raise and lower arms
6 Hold arms out, completely bend and straighten elbows
7 Hold arms down, bend elbows 90° and pronate and supinate forearm
8 Make a fist, clench and then spread the fingers
9 Squat and duck-walk 4 steps toward you
10 Stand straight with arms at sides, back to you
11 Bend forward with knees straight and touch toes
12 Stand on heels and rise to the toes

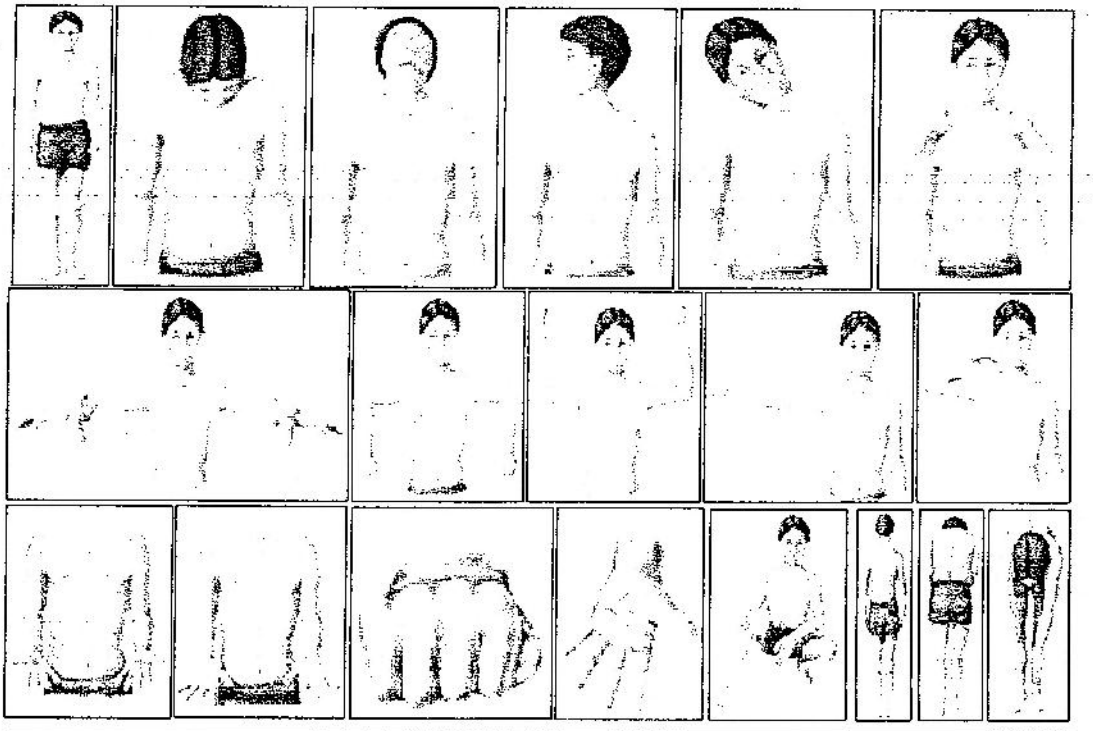


FIGURE 16 – 4. Screening tests of MSK system examination.

NEONATAL HISTORY

As that of older patients, the guidelines are the same. Remember that there is no need to request past history or developmental history. The following steps are suggested:

1 PERSONAL HISTORY
2 CHIEF COMPLAIN AND IT'S DURATION
3 HISTORY OF PRESTENT ILLNESS
4 The history of present illness consists of the following axes:
   Analysis of chief complaint
   Birth history (usually post natal history)
   Admission, investigation and treatment
5 REVIEW OF OTHER SYSTEMS
6 FAMILY HISTORY
7 SOCIOECONOMIC HISTORY
8 VACCINATION HISTORY
9 FEEDING HISTORY
10 DRUG HISTORY
11 Stand on heels and rise to the toes

EXAMPLE

Zaineb is a product of a normal vaginal delivery in hospital at 8 months age and she is a member of twin. Her mother is 25 years old, G_2P_2, her blood group is O+ with good health during pregnancy. She is not diabetic nor hypertensive, with poor ANC. 1 week before labor, the mother had history of leaking liquor with fever. She had delivered at hospital and the product was twin, one of them unfortunately died immediately and the second one started respiration after resuscitation. She was started then to complain of SOB, decreased activity and failure to feed. At the 3rd day, the baby developed a yellowish discoloration of skin which progressed then to involve the whole body skin. The family then consulted their pediatrician to.....

NEONATAL EXAMINATION

All babies, if possible, should be examined immediately at birth then after 24 hours and finally at the end of the first week. The examination of the newborn should be performed in front of his/her mother. The mother, of course, should be involved in all stages of examination. The examiner's duty is to explain to the mother what is he/she doing.
In neonate, it is regional examination from the head to the toes as follows:

GENERAL LOOK AT THE NEWBORN

Look to the newborn and determine whether he/she is ill or well and then examine for jaundice, anemia, cyanosis, edema, and skin lesions. Jaundice that appears within the first 24 hours of birth is likely to be pathologic jaundice due to hemolytic disease of the newborn. Jaundice that persists beyond 2 – 3 weeks should raise suspicions of biliary obstruction or liver disease. Jaundice can best be appreciated in natural daylight rather than artificial light. Newborn jaundice seems to progress from the head to the toe, with more intense jaundice on the upper body and less intense yellow color in the lower extremities. To detect jaundice, apply pressure to the skin as shown in FIGURE 16 – 5 to press the normal pink or brown color out. Look for the presence of a yellowish blanching, which indicates jaundice.

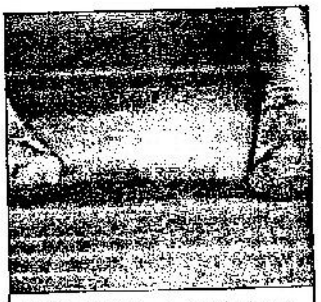


FIGURE 16 – 5. Technique to check for jaundice.

Vasomotor changes in the dermis and subcutaneous tissue a response to cooling or chronic exposure to radiant heat can produce a lattice-like, bluish mottled appearance called cutis marmorata particularly on the trunk, arms, and legs. This response to cold may last for months in normal infants. Cutis marmorata is prominent in premature infants, and in infant with congenital hypothyroidism and Down syndrome. Acrocyanosis: a blue cast to the hands and feet when exposed to cold, is very

common in newborns for the first few days, and may recur throughout early infancy. If acrocyanosis does not disappear within 8 hours or with warming, cyanotic congenital heart disease should be considered. Occasionally in newborns, a striking color change harlequin dyschromia appears with transient cyanosis of one half of the body or one extremity, presumably due to temporary vascular instability. A dark or bluish pigmentation over the buttocks and lower lumbar regions is common in newborns of African, Asian, and Mediterranean descent. These areas, formerly called Mongolian spots, are due to the presence of pigmented cells in the deep layers of the skin; they become less noticeable with age and usually disappear during childhood. It is important to document these areas to avoid later concern about bruising. Central cyanosis in a baby or child of any age should raise suspicion of congenital heart disease .The best area to look for central cyanosis is the tongue and oral mucosa, not the nail beds or the extremities. At birth there is a fine, downy growth of hair called lanugo over the entire body, but especially the shoulders and back. This hair is shed within the first few weeks. Lanugo is prominent in premature infants. Hair thickness on the head varies considerably among newborns, and fortunately is not predictive of later hair growth. All of the original hair is shed within months, replaced with a new crop, sometimes of a different color.

You should be able to identify four common dermatologic conditions in newborns. None is clinically significant. Milia, pinhead-sized smooth, white raised areas without surrounding erythema on the nose, chin, and forehead, are caused by retention of sebum in the openings of the sebaceous glands. While occasionally present at birth, milia usually appear within the first few weeks and disappear over several weeks. Miliaria rubra consists of scattered vesicles on an erythematous base, usually on the face and trunk, caused by sweat gland duct obstruction; it disappears within weeks spontaneously. Erythema toxicum, which usually appears on the 2nd or 3rd day of life, consists of erythematous macules with central pinpoint vesicles scattered diffusely over the entire body, appearing much like flea bites. These lesions are of unknown etiology, but disappear within a week after birth. Pustular melanosis, seen more commonly in black infants, is present at birth as small vesiculopustules over a brown macular base and can last for several months. Both erythema toxicum and pustular melanosis may appear similar to the pathologic vesicular rash of herpes simplex or staphylococcus aureus skin infection, which are serious infections requiring rapid treatment. Midline hair tufts over the lumbosacral spine region suggest a spinal cord defect.

HEAD

Check the shape and size; skull (fontanelles, sutures and bones) and scalp defect or other abnormalities shown in BOX 16 – 32.

BOX 16 – 32. Comparison between caput succedaneum and cephalohematoma

Caput succedaneum	Cephalohematoma
Edema	Subperiostal bleeding
Exceeded sutures	Limited to the bone
Disappears within days	Disappears within 4 – 6 weeks
No jaundice or anemia	Jaundice and anemia

THE EYES

Held the baby from occiput with your left hand and shake him/her. Microphthalmia may be seen in hypothyroidism. Bophthalmos may occur in congenital glaucoma. Opacity of the lens may occur in TORSCH infection.

THE EARS

Check their shape; normal or low seated and then check patency of external auditory meatus.

THE NOSE

Try to check the nasal bridge. The check both nostrils and ascertain whether they are opened (patent) or not.

THE MOUTH

Check for cleft lip or palate; check for any abnormalities in the tongue; check the for natal teeth.

THE NECK

Look for central masses as goiter or hypoglossal cyst, lateral masses as cystic hygroma, branchial cyst and sternomastoid hematoma or tumour.

THE CHEST

Check shape of the chest; signs of dyspnoea should be sought. For the lung, auscultate two areas bilaterally and axillae to detect air entry and respiratory rate. Auscultate also from the back. For the heart, auscultate two areas, one over the nipple, and the other is below it checking for S1 and S2 and murmur.

THE ABDOMEN

Liver and spleen are palpated using two fingers; kidneys are bimanually palpated. Check for ambiguous genitalia, hypospadius or epispadius and for undescended testis.

THE ANUS
Check if patent or imperforated.

THE BACK
Check for meningocele, meningomyelocel and encephalocele.

FEMORAL PULSES
Extend knee joints and put the fingers of both hands on popliteal areas of both limbs. Each thumb is placed on mid-inguinal point exactly on femoral arteries.

CONGENITAL DEVELOPMENTAL DISLOCATION OF THE HIP
Use the following two tests (FIGURE 16 – 6):

1 Ortolani test

Place the baby supine with the legs pointing toward you. Flex the legs to form right angles at the hips and knees, placing your index fingers over the greater trochanter of each femur and your thumbs over the lesser trochanters. Abduct both hips simultaneously until the lateral aspect of each knee touches the examining table. A palpable movement of the femoral head back into place constitutes a positive Ortolani sign. With a hip dysplasia, you feel a clunk as the femoral head, which lies posterior to the acetabulum, enters the acetabulum

2 Barlow test

Place your hands in the same position as for the Ortolani test. This time, press in the opposite direction with your thumbs moving down toward the table and outward. Feel for any movement of the head of the femur laterally. Normally there is no movement and the hip feels stable. If you feel the head of the femur slipping out onto the posterior lip of the acetabulum, this constitutes a positive Barlow's sign. If you do feel this dislocation movement, abduct the hip by pressing with your index and middle fingers back inward and feel for the movement of the femoral head as it returns to the hip socket. A positive Barlow's sign is not di-agnostic of a dysplastic hip, but it indicates laxity and a dislocatable hip progressively, and the baby needs to be reexamined in the future.

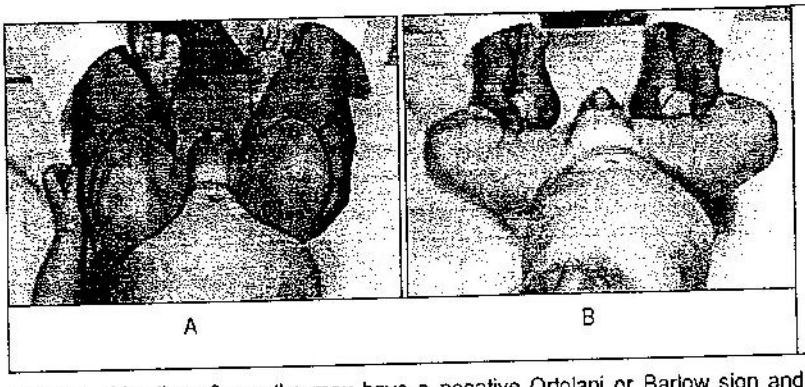


FIGURE 16 – 6. Ortolani's test (A) and Barlow test (B).

Children older than 3 months may have a negative Ortolani or Barlow sign and still have a dislocated hip due to tightening of the hip muscles and ligaments.

THE LIMBS
Check for absent or extra-digits and edema at the dorsum of the foot.

NEUROLOGICAL REFLEXES
These primitive reflexes should be part of the routine neurologic examination of infants. Neonatal reflexes are weak or even absent in premature infants. Some of these tests can be elicited since birth whilst others appear later. These are:

1 Moro reflex (FIGURE 16 – 7)

With the baby supported in a horizontal position by your left hand, the head is lifted by palm of your right hand placed under back of neck to 2-3 cm from the horizon. The right hand is now released rapidly and a sudden movement of the cervical region produces a reflex response. This consists of abduction and extension of the arms with the hands opened followed by adduction of the arms across the chest

2 Rooting reflex (FIGURE 16 – 7)

When the corner of the infant's mouth is lightly touched, the lips and tongue deviate towards the tongue in an attempt to find the nipple and take it into the mouth. The head of the infant may also turn towards the touch

3 Sucking reflex

The little finger is introduced into the mouth touching the hard palate and also palpate for cleft palate

4 Grasp reflex (Palmer and Planter reflex) (FIGURE 16 – 7)

A finger placed across the palm will cause a flexion and grasping of fingers. A similar response is seen if a finger is placed on the planter surface of the foot

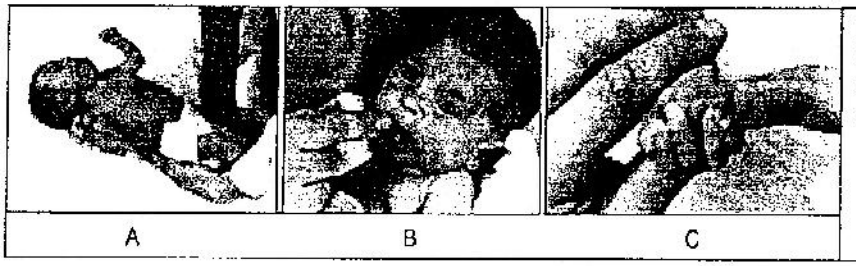


FIGURE 16 – 7. Primitive reflexes including Moro (A), rooting (B), and grasping (C) reflexes.

5 Placing reflex (FIGURE 16 – 8)

The infants place foot on examining surface when the dorsum of the foot is brought in contact with edge of the surface

6 Walking reflex or stepping reflex (FIGURE 16 – 8)

The baby is held upright and the feet placed on a firm surface as the foot presses down on the surface, the other leg flexes at the hip and knee in a stepping movement. As this response is alternated from one to other, the baby makes a walking movement

7 Crossed extension reflex

This reflex is obtained by holding one leg in extension and by stimulating the sole of the same foot. This elicits flexion of the opposite leg followed by adduction and then extension

8 Tonic neck reflex (FIGURE 16 – 8)

When the baby lies in supine position, turn the head to one side. This is followed by extension of the arm and legs on the side to which the head is turned whilst the opposite knee and elbow are flexed

9 Glabellar tap

When the infant's eyes are opened a gentle tap of the forehead between the eye brows will elicit a blink reflex

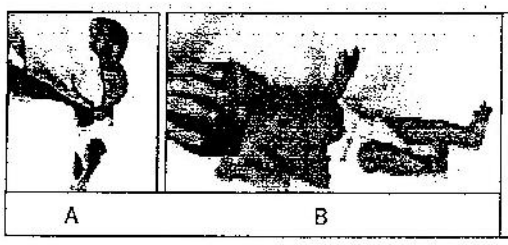


FIGURE 16 – 8. Primitive reflexes including placing and walking reflex (A) and tonic neck reflex (B).

PRIMITIVE REFLEXES APPEARING LATER

1 Trunk incurvation reflex (FIGURE 16 – 9)

The infant withdraws from stoking stimulus along the ventral margin

2 The parachute reflex (FIGURE 16 – 9)

With the infant sitting, tilting to either sides results in extension of the ipsilateral arm in protective fashion. This reflex starts at 6 – 8 months and remains for life

3 Landau reflex (FIGURE 16 – 9)

With the infant held about the waist, suspended extension of the neck causes extension of the arms and legs. This reflex starts at 6 – 8 months and disappears at 15 month to 2 years

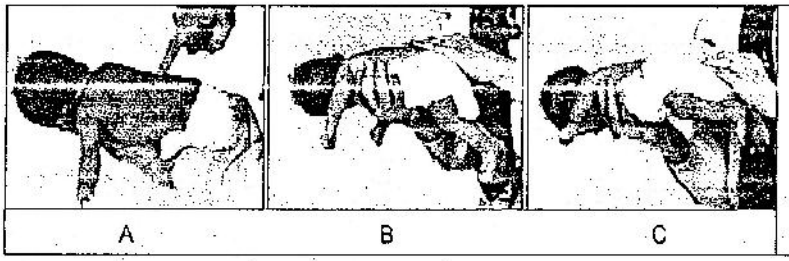
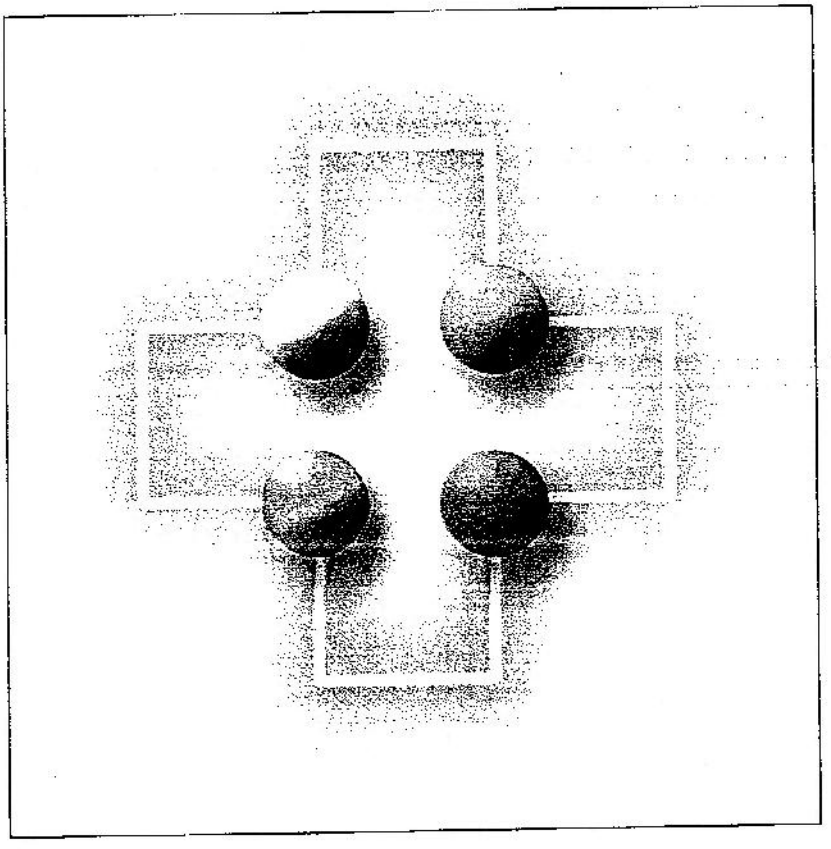


FIGURE 16 – 9. Primitive reflexes appearing later including trunk incurvation (A), parachute reflex (B), and Landau reflex (C).

17

LAB RESULTS



LIST OF NORMAL VALUES FOR THE COMMON CLINICAL BIOCHEMICAL AND HAEMATOLOGICAL TESTS IN ADULT

TEST	NORMAL VALUE (TU)	CONV. F (TSI)	NORMAL VALUE (SIU)	CONV. F (SIT)
BIOCHEMISTRY				
S. ALBUMIN	3.6 – 5.2 g/dl	10	36 – 52 g/l	0.1
S. ACID PHOSPHATASE	0 – 0.8 U/l	1	0 – 0.8 U/l	1
S. ALKALINE PHOSPHATASE	30 – 85 U/l	1	30 – 85 U/l	1
S. ALT (SGPT)	< 20 U/l	1	< 20 U/l	1
S. AMYLASE	23 – 85 U/l	1	23 – 85 U/l	1
S. AST(SGOT)	< 15 U/l	1	< 15 U/l	1
S. TOTAL BILIRUBIN	0.3 – 1.0 mg/dl	17.1	5 – 17 micromol/l	0.06
S. CALCIUM (total)	8.5 – 10.5 mg/dl	0. 25	2.1 – 2.6 mmol/l	4
U. CALCIUM	100 – 350 mg/day	0.25	25 – 87 mmol/day	4
S. CHLORIDE	95 – 105 mEq/l	1	95 – 105 mmol/l	1
S. CHOLESTEROL	150 – 250 mg/dl	0.0259	3.87 – 6.47 mmol/l	38.7
S. COPPER	80 – 150 microgram/dl	0.1625	13 – 24 micromol/l	6.15
U. COPPER	10 – 50 microgram/dl	0.1625	1.6 – 8.1macromol/l	6.15
S. CORTISOL (morning)	7.14 – 23.2 microgram/dl	28	200 – 650 nanomol/l	÷28
S. CORTISOL (night)	<300 micrgram/dl	28	< 10.7 nanomol/l	÷28
S. CREATININE KINASE	20 – 230 U /l	1	20 – 230 U/l	1
S. CREATININE	0.7 – 1.4 mg/dl	88.4	62 – 124 micromol/l	0.0113
U. CREATININE	1.0 – 2.0 g/day	8.84	9 – 17 mmol/day	0.116
S. GAMMA G.T	5 – 85 U/l	1	5 – 85 U/l	1
S. GLUCOSE (fasting)	65 – 110 mg/dl	0.0555	3.6 – 6.1 mmol/l	18
CSF. GLUCOSE	40 – 70 mg/dl	0.0555	2.2 – 3.8 mmol/l	18
U.HMMA (VMA)	< 8 mg/day	5.05	<40 micromol/day	0.2
S. IRON	70 – 180 microgram/dl	0.179	13 – 32 micromol/l	5.62
S. TIBC	250 – 400 microgram/dl	0.179	45 – 70 micromol/l	5.62
S. MAGNESIUM	1.8 – 2.4 mg/dl	0.411	0.7 – 1.0 mmol/l	2.4
U. MAGNESIUM	80 – 120 mg/day	0. 411	33 – 50 mmol/day	2.4
S.INORGANIC PHOSPHATE	2.5 – 4.5 mg/dl	0.323	0.8 – 1.4 mmol/l	3.1
S. POTASSIUM	3.5 – 5.3 mEq/l	1	3.5 – 5.3 mmol/l	1
PROGESTERONE (males)	0.13 – 1.26 nanogram/ml	3.17	0.4 – 4.0nanomol/l	0.315
PROGESTERON (females)				
Follicular peak	0.06 – 10.26 nanogram/ml	3.17	0.2 – 4.0 nanomol/l	0.315
Ovuteal phase	0.08 – 1.2 nanogram /ml	3.17	0.25 – 3.8 nanomol/l	0.315
Luteal phase	2.5 – 25 nanogram/ml	3.17	8 – 78 nanomol/l	0.315
S. TOTAL PROTEIN	6.2 – 8.2 g/dl	10	62 – 82 g/l	0.1
S. GLOBULIN	2.4 – 3.7 g/dl	10	24 – 37 g/l	0.1
CSF-PROTEIN	15 – 40 mg/dl	0. 01	0.15 – 0.4 g/l	100
U. PROTEIN	<100 mg/day	1	<100 mg/day	1
S. SODIUM	136 – 155 mEq/l	1	136 – 155 mmol/l	1
TESTOSTERONE(males)	2.8 – 8.2 nanogram/ml	3.46	9.7 – 28.4 nanomol/l	0.289
TESTOSTERONE(females)	0.1 – 1.0 mamogram /ml	3.46	0.34 – 3.4 nanomol/l	0.289
THYROXIN(T4)	4.5 – 12 microgram/dl	12. 9	60 – 155 nanomol/l	0.0775

CONTINUED				
TRIIODOTHYRONIN(T3)	0.8 – 1.9 nanogram/ml	1. 5	1.2 – 2.85 nanomol/l	÷1.5
S.TRIGLYCERIDES	65 – 180 mg/dl	0. 0133	0.86 – 2.4 mmol/l	75.3
S.URIC ACID	3.0 – 7.0 mg/dl	60	180 – 420 mmol/l	0.0166
U.URIC ACID	250 - 750 mg/day	0.06	1.5 - 4.5 mmol/day	16.6
S.UREA	20 – 45 mg/dl	0.166	3.3 – 7.5 mmol/l	6
U.UREA	15 – 30 g/day	16.6	250 – 500 mmol/day	0.06
IMMUNOGLOBULINS				
IgG	700 – 1800 mg/dl	0.01	0.9 – 4.5 g/l	100
IgM	50 – 220 mg/dl	0.01	0.5 – 2.2 g/l	100
HEMATOLOGY				
Hb -men	12 – 18 g/dl	10	120 – 180 g/l	0.1
Hb -women	11.5 – 16.5 g/dl	10	115 – 165 g/l	0.1
PCV	0.4 – 0.45%	100	40 – 45%	0.01
MCHC	31 – 35 g/dl	10	310 – 350 g/l	0.1
WBC	4000 – 11000/mm^3	0.001	4 – 11 X 10^9/l	1000
PLATELETS	150000-400000/mm^3	0.001	150 – 400 X 10^9/l	1000